



## METHODS IN MEDICINE



# Methods in Medicine

THE MANUAL of the MEDICAL SERVICE  
of

GEORGE DOCK, M D, Sc.D

Formerly Professor of Medicine Washington University School of Medicine  
Formerly Physician in Chief Robert A. Barns Hospital St. Louis

A Comprehensive Outline for Clinical Investigation,  
Management, and Treatment of Patients  
with Various Medical Disorders

by

GEORGE R. HERRMANN, M D, Ph D

Professor of Medicine University of Texas Medical Branch at Galveston Director of  
the Cardiovascular Service and Heart Station University of Texas Hospitals  
Consultant in Medicine to the Surgeon General U S Army Consultant in  
Vascular Diseases to the Marine Hospital U S P H S

*Second Edition Completely Revised*

St. Louis  
THE C. V. MOSBY COMPANY  
1950



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Printed in the  
United States of America

*Press of*  
*The C V Mosby Company*  
*St Louis*

TO  
GEORGE DOCK  
MASTER CLINICIAN  
AS A  
FESTSCHRIFT  
IN HONOR OF  
HIS NINETIETH BIRTHDAY  
APRIL 1 1950



## PREFACE TO SECOND EDITION

A quarter of a century has elapsed since the first edition of this manual was published at the close of the great medical service of Dr George Dock. The manual embodied an efficient system of organization which proved successful and contained an expanded version of many of the ideas of the 'Precedent Book of the Medical Service' of Professor Henry A Christian. Many of the present day university medical services have been founded on these same precedents which probably originated in Professor William Osler's medical services at the University of Pennsylvania and at the Johns Hopkins University Hospitals in which my chiefs had served. It seemed fitting to reissue the manual brought abreast with the times as a Festschrift in honor of the ninetieth birthday of Dr George Dock sixty years after the opening of Dr Dock's first laboratory at the University of Texas and just as a new University of Texas service is being organized at Dallas.

In the twenty six years since the first edition of this manual came off the press the science of internal medicine has been greatly advanced by a tremendous volume of research and many discoveries in diagnostic methods as well as therapeutic agents. Insulin liver extracts vitamins hormones sulfonamides antibiotics and a host of synthetic organic drugs have been produced and have been great boons to suffering humanity. The advances in methodology have not been so spectacular and the organization plans have changed only in the addition of more subspecialties as hematology endocrinology nutrition allergy physiology cardiology, gastro enterology hepatology and angiology all of these still come under internal medicine.

The manual has been planned as a practical ward or bedside guide for the clinical investigation of the common and some of the more rare conditions. Sound minimal requirements for the systematic diagnostic study case recording emergency case study and handling, scientific medical management and dietetic therapy are outlined in succeeding chapters. Each method has been selected that has been tried and proved to be satisfactory. The methods are logically arranged and condensed but with the retention of sufficient details for the execution of the method and the general interpretation of the results. The intern the resident, and the practitioner it is hoped will find in this little volume, the methods he needs in solving his usual medical diagnosis and therapeutic problems.

The table of contents is set up in detail to serve as an outline or guide for clinical and laboratory procedure. The sources of material and references are set down whenever it is possible to do so.

I am deeply indebted to many besides my chiefs, my associates, and student assistants, to my wife and my children without whose loyalty, inspiration, and unmitigated and constant help I could not have completed this effort. My daughters, Georgeanna H Greer, M D, Gretchen H Runge, M D, my son, George D H C Herrmann, M D, and my wife, Anna H Williams, gave me constant support and stimulating encouragement throughout this effort. My thanks are due to my chief associate, Dr Milton R Hejtmancik, who carefully worked over the proofs with me.

GEORGE R HERRMANN

Galveston, Texas

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## PART III

### METHODS OF CLINICAL INVESTIGATION

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## CHAPTER I

### SORTING OUT THE VASCULAR DISORDERS OF THE LIMBS

ARTERIAL deficiency in an extremity is obviously unfavorable to its use. Minor deficiencies cause functional disorders. Serious ones lead to malnutrition and even necrosis. If arterial stoppage develops slowly, the natural sequence of events is coldness, atrophy, and finally gangrene—a dry, shrinking, and only slowly destructive process. The first external sign of this change in the legs is a slowing of locomotion. The individual steps out, fails to obtain a sufficient blood supply for his laboring muscles, rests and sets out again, suffering in fact the intermittent claudication first described a hundred years ago, in the case of the horse, by the French veterinarians. In other respects, save for a lack of resistance to cold, there is little sign of anything wrong. Acute stoppage, on the other hand, is spontaneously paralyzing and usually painful. The individual is struck down and can not use the affected limb. Incidentally, since the tissues are not gradually drained but caught wet, as it were, any gangrene which occurs is apt to be moist and far more liable to infection than the dry, mummifying variety. One particular sort of deficiency, due to the somewhat premature arteriosclerosis of diabetes, is in the main of slow onset, though owing to the diabetic's lack of resistance to infection, acute emergencies often arise.

The venous and lymphatic disorders are, by contrast, causes of congestion, of heaviness. They do not disable unless the limb becomes mechanically unwieldy or, losing its resistance to injury and infection, becomes chronically inflamed or ulcerated. To induce swelling of a limb, the obstruction in the returning circulation must be of a high grade, the principal vessels must be occluded and probably over a considerable



area. Thus thrombosis of the iliac and femoral veins causes enlargement of the entire leg, whereas a failure of the superficial venous return, as in varix, and even an extensive thrombosis of varicose veins, leads to almost no swelling.

Such gross distinctions as these are clear enough, and if all circulatory disorders fell into these categories, they would very readily be understood. Unfortunately there are arterial diseases which are not organic but functional, not permanent but spasmodic—disorders of vasomotor control, whether through the sympathetic system or chemical mediation, unnatural reactions to the emotions and especially to cold, such as Raynaud's disease. There are, also, combinations of arterial and venous occlusion, notably thromboangiitis obliterans (Buerger's disease). There are arterial spasms apparently reflex in nature and secondary to a variety of wounds and injuries. Some of these are temporary, others are prolonged and associated with serious changes in skin, bones, and joints—painful states, as a rule, including the causalgias. There are even arterial spasms secondary to, or at least associated with, venous thromboses. Finally, there are inexplicable and permanent states of vasomotor irritation causing cyanosis, coldness, sweating, and, in some instances, ulceration of the extremities.

The arterial aneurysms and venous malformations, such as nevi and the arteriovenous fistulas both congenital and traumatic, also affect the limbs, though since they exhibit themselves in other places they are by no means peculiar to these parts. A brief account of them, if only for purposes of comparison, will therefore be included here.

**The Arms and the Legs**—There is a distinction of some importance, rather helpful in diagnosis, between the diseases of the arms and the legs. Whether because the blood pressure is higher in the lower limbs than the upper or because of the wear and tear due to a more violent use, the legs commonly show the effect of arteriosclerotic stiffening and endarteritis, whereas the arms almost never do so. This is true both of the chronic narrowings and acute thromboses. The combined

thrombosis of both arteries and veins in thrombo angitis obliterans is also far more troublesome to the legs than the arms, though here the distinction is much less clean cut than it is in the case of the purely arteriosclerotic disorders. The legs and pelvis are also the seat of most of the venous thromboses and of the unusual secondary arterial spasms related to them.

The arms, by contrast, tend to be the usual site of the many states of arterial spasm brought about through the mediation of the vasomotor government in general. To all generally distributed reactions the arteries of the legs of course respond, though in a less noticeable and troublesome way than do those of the arms. But there are certain states, such as the rare arteritis, associated with irritation of the brachial plexus by a cervical or first rib, and the effects of certain injuries of the great nerves, causalgia or causalgia like states, which are almost peculiar to the upper extremities.

There is also a distinction between states of spasm and organic disease, not to be taken too seriously but helpful in diagnosis. The area principally subject to the effects of peripheral arterio spasm occupies little more than the feet and hands. That is to say, it is limited centrally and sometimes with almost a glove or shoe like abruptness shortly above the wrist and the ankle. Thus the diseases which show themselves in changes confined to such areas will most often have a predominantly vasospastic background. See, for instance, a youngish man, a heavy smoker, suffering from a pronounced intermittent limp and find, on passing your hand down his legs, that cool skin is encountered rather abruptly at almost the level of the shoe tops. Whatever degree of organic obliteration he may exhibit, you may properly judge that there is also an element of sympathetically controlled arterial spasm in his case.

This rough analysis of the vascular diseases of the extremities will serve as the outline for this book. Information about them is increasing and at such a pace that classification is apt to be inadequate and explanation fallacious. However, there is here outlined a sort of index of disabilities which may be

useful in pigeonholing the various circulatory disorders

Arteriosclerotic Deficiencies

Diabetic Infections and Gangrene

Thrombo-angitis Obliterans

Vasospastic Disorders

Raynaud's Phenomenon

Vasospasms, acute traumatic

Vasospasms, chronic and related to injury and infection

Vasospasms, functional, permanent and unclassifiable

Arterial Embolism

Varicose Veins and Ulcer

Thrombophlebitis and Venous Embolism

Post phlebitic States

Arterial Aneurysm and Abnormal Arteriovenous Communications

Lymphedema, Lymphangioma and Elephantiasis

The most significant function of the circulation is the nourishment of the tissues. At first sight it might seem that this function, in the case of the limbs, is quite the same as that performed for the rest of the body. Such is hardly the case. Though it is true that when at rest the limbs make little demand upon the circulation, in action their demands are enormous. Everyone is familiar with the slowing up of the athlete, the giving out of his legs, in the late thirties. His heart is probably as good as ever. His endurance, partly owing to his increased experience and skill, is probably greater. But his elasticity, as opposed to his capacity for sustained effort, that is, his capacity for muscular response at high speed, is beginning to lessen. The cause of this change lies presumably in the failure of his arterial system to respond actively enough to the call made upon it by the muscles. Carry this functional deficiency a little farther and you have a basis for the intermittent limp. At rest, the legs of the individual whose arterial system is becoming limited are comfortable. In action, they are insufficiently supplied with blood, and the characteristic intermittent painful numbness sets in. Carry the circulatory

deficiency still farther, combine it with minor injuries and the cooling of small parts with large surfaces, and the extremities become more than functionally ill nourished. They are actually liable to necrosis—senile or presenile gangrene.

The second function, and one upon which familiar observations and tests depend, is the maintenance of the surface temperature. This of course is only a part of the control of body temperature in general. However, the local cutaneous temperature is of chief interest here. Warmth of the skin is a sign of an abundant blood supply. Coldness is an evidence of a deficient circulation. This is simple enough. But a deficient circulation may be restricted either organically or merely by functional contraction. And so a distinction must be made between these two states, a matter of deciding whether or not the arterial system, especially the finer parts of it, are capable of contraction and expansion. An organically deficient arterial supply makes a relatively cold surface but above all is so inelastic as no longer to respond to a reflex call for temperature regulation. Thus the skin of a limb tends to take on, not the deep temperature of the body but the temperature of its environment. It can slowly be warmed by the surrounding air, and rather more readily be cooled. A normal set of vessels responds to a hot environment, and particularly to any signal received from the heat regulating centers, by dilatation with consequent warming and flushing of the skin in an effort to disseminate heat, the medullary centers being extremely sensitive to *any* rise in the temperature of the blood. By the same token, normal vessels respond to external cooling by contraction, lest heat be lost through radiation. Now just as stiff, contracted arteries fail to respond to such influences, unnaturally irritable vessels may respond too readily. A little cooling of the environment, a little nervousness or fear, sends them into a state of spasm, whereby the skin becomes pale (or blue) with cold. This is of course a gross picture. The normally and overly responsive small arteries are spoken of as if their reactions were only to nervous control of body temperature in general. As a matter of fact they respond to local stimuli

as well. By warming a foot, for instance, its small vessels can be made to dilate quite apart from any general influence.

The large and small vessels do not react, respectively, in quite the same way to contracting and dilating influences. Vasodilatation and vasoconstriction chiefly occur in the small arteries and arterioles. Thus it is upon the surface, particularly in the feet and hands and above all in the fingers and toes, that changes in surface temperature are most sensitively displayed. Sir Thomas Lewis has revealed the purpose of the numerous fine arteriovenous anastomoses, muscular and richly innervated, which are present upon the surface of the body and especially in the digits, the palms and the soles. These, when dilated, permit a flood of arterial blood to warm the skin. Similarly, their contraction aids in cooling the surface. Thus, as compared with the body and the more bulky part of the limbs, the feet and hands are far more rapidly warmed and cooled and quickly reveal their state in the temperature of the skin. It is, in fact, in the fingers and toes that peripheral vasoconstriction and vasodilatation can most easily be studied.

The Sympathetic System, previously mentioned but not actually described, now requires consideration. It is an out-flowing mechanism, exercising vascular control and, by the stimulus of pain, rage and fear, energizing the body for action. Shakespeare might have been collaborating with Walter Cannon when he wrote

“In peace there’s nothing so becomes a man  
As modest stillness and humility  
But when the blast of war blows in our ears,  
Then imitate the action of the tiger  
Stiffen the sinews, summon up the blood,  
Disguise fair nature with hard favored rage  
Then lend the eye a terrible aspect,  
Let it pry through the portage of the head,  
Like the brass cannon, \*\*\*  
Now set the teeth, and stretch the nostril wide,  
Hold hard the breath, and bend up every spirit  
To his full height!”

*Henry V Act III, Scene 1*

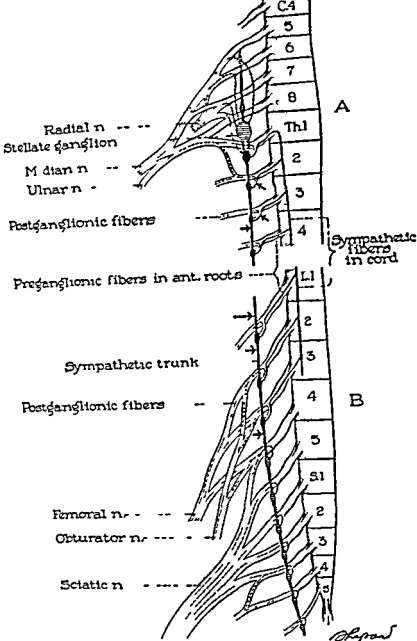


FIGURE 1. A DIAGRAMMATIC SKETCH OF THE SYMPATHETIC INNERVATION OF THE LIMBS. The sympathetic ganglia and chain, solid black; the preganglionic rami, solid black; the postganglionic rami, dotted lines. Arrows indicate points at which the rami and chain should be interrupted to secure preganglionic denervation. Interruption above L<sub>1</sub> and removal of L<sub>1</sub>, 2 and 3 gives vasodilatation as high as the thigh but is only to be practiced in females.

In other words, the sympathetic system, stimulated by any excitement, raises the blood pressure, constricting the arteries, and drives the blood to the muscles which it abundantly furnishes with immediately usable fuel. But in the meanwhile, it stops all processes not immediately required for action. By causing the peripheral vessels to contract, it halts all loss of heat, at the same time turning the surface pale, dilating the pupil and widening the eye, and by an action usually but not always consistent with this vasomotor response, it erects the hairs and sets the skin to sweating. By contrast, paralysis of the sympathetic leaves the muscular arteries relaxed, the skin flushed and dry, the pupils contracted. In the normal, balanced state, the sympathetic, of course, maintains a steady vasomotor tone.

All this is accomplished by a system consisting of a series of nerve cells and relay stations. From cells in the very oldest part of the brain, fibers descend the cord and pass out with the anterior spinal roots to make contact with secondary cells in the long, paravertebral ganglionated chain. The little nerves which carry these fibers from the cord to the ganglions are the preganglionic or white rami. Through every one of these, several ganglia are activated. Next, from the cells in the ganglia, other fibers pass on to be distributed via the peripheral nerves to the blood vessels and sweat glands all over the body. These are the gray or postganglionic rami. There are other way stations to the various thoracic and abdominal viscera, but the simple system just described serves for the blood vessels. In Figure 2 the distribution of sympathetic fibers to the arterial system of the limbs is pictured. It will be noticed that the fibers flow out upon the great vessels from the principal nerves at a series of levels but do not travel far along them, and that the distribution of the sympathetic supply from the principal nerves of each limb corresponds to their respective sensory fields upon the surface.

To this description it is only necessary to add that the sympathetic supply to the arm arises in the upper thoracic cord, passing out by white rami to the second and third thoracic

ganglia and along the sympathetic chain to the stellate ganglion (first thoracic and inferior cervical), thence, principally from cells in the stellate, in gray ram, to the brachial plexus. The supply to the leg comes from the lower thoracic and lum

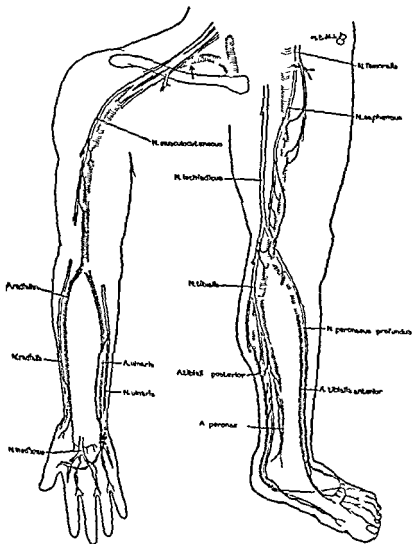


FIGURE 2 THE DISTRIBUTION OF SYMPATHETIC NEURONS TO THE PERIPHERAL BLOOD VESSELS (semidiagrammatic and incomplete—after Kramer and Todd and Potts) The arrow indicates the point at which fibers destined for the brachial artery may be exposed to irritation as they pass over the highest rib



bar cord Some preganglionic fibers pass in white rami to the second and third lumbar ganglia, and from these ganglia a number of postganglionic fibers go out to the vessels of the thigh But most of the preganglionic fibers descend in the sympathetic chain to ganglia below the second and third lumbar, where they make their contact with cells whose postganglionic fibers are distributed, via the sciatic to the principal arterial system of the legs This arrangement permits division of the preganglionic rami to both upper and lower limbs without injury to the cells of the postganglionic fibers Thus, stimuli from the central nervous system can be cut off\* from the vessels of the limbs without loss of the *local* postganglionic governing mechanism This feature will be developed in the story of the operative treatment of vasospasm (Chapter IV) and is a fundamental consideration in present-day vascular surgery

In the foregoing paragraphs the vasomotor nerves have been described as if they consisted only of vasoconstricting fibers About outgoing vasodilating fibers, if such exist,† less seems to be known Vasodilatation takes place not only when central vasoconstrictor control is artificially lifted but under the influence of the products of local metabolism which cause the blood supply to fluctuate very delicately and accurately according to the needs of the tissues The blood supply of the muscles is governed in this way If the circulation to a given area of muscle is arrested, sufficient vasodilatation occurs in

\* It is confusing to discover as Oughterson, Harvey and Richter have done, that after an apparently complete sympathetic denervation of the lower limb some vasomotor control yet remains (posterior tibial nerve block causes additional vasodilatation) Their experimental observations suggest that vasoconstrictor fibers reach the sciatic nerve from sources in the spinal cord lower than the lowest hitherto recognized exits for sympathetic nerves (that is caudad to the third and even the fourth lumbar roots)

† In the normal individual they have never been proved to exist However, Lewis and Pickering have shown that in anyone suffering from vasospasm as in Raynaud's disease full vasodilatation in the little finger as called forth by heat is prevented by blocking with procaine the corresponding ulnar nerve Yet blocking the ulnar nerve abolishes all vasoconstriction in the ulnar field paralyzing vasoconstriction fibers Thus it would seem also to paralyze vasodilator fibers A parallel phenomenon has been found true in the leg

that area so that when blood flow is again released its volume is greatly increased until the oxygen debt is paid

The statement has also been made that sympathetic nerves do not travel for any great distance upon large arteries. This does not deny the fact that stimuli of some sort—possibly in going sensory impulses, for arteries appear to be sensitive—do travel along the great vessels. The fixation of an embolus in the femoral artery, for instance, or an inflammatory reaction in its wall may be associated with peripheral spasm in the area served by the artery. Cut out a portion of this great vessel, which Leriche likens to a long, inflamed nerve, and a vicious reflex cause of vasospasm is at once relieved, a reaction which usually improves the circulation in the peripheral field. Whether that division has succeeded because of interrupting in going or out going impulses is still a question. Apparently thrombosed or inflamed veins occasionally excite a similar reflex vasospasm. Injured nerves are capable, under unusual circumstances, of setting up a somewhat similar symptom complex. That such states involve in some way the sympathetic system is beginning to be made clear by the relief secured from a temporary or permanent block cutting off all the sympathetic impulses to the part affected. However, as will appear when these states are discussed, the mechanism in most instances is not only difficult to understand but is not necessarily of the same sort in different cases. All that can be affirmed is that a persistent vasospasm, even of a large arterial tree, can take place as a result of a considerable variety of stimuli affecting not only the arteries themselves but the great veins and nerves as well.

**Vascular Exercise and Reactive Hyperemia.**—In the previous paragraphs some account has been given of the normal and pathological physiology of vasoconstriction in so far as it affects the limbs, and the suggestion has been made that vasodilatation, the reverse of that picture, occurs, not as an independent process, but as one which takes place when vasoconstriction is abolished, as by fever or as a reaction after temporary arrest of the circulation. That is, vasodilatation

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in his historical account of the procedure And indeed Bier seems to have used venous hyperemia primarily to cure acute and chronic infections and not to influence the circulation alone However, he established the facts (1) that venous hyperemia induces a reactive hyperemia independently of nervous impulses, (2) that prolonged moderate venous stasis (many will remember their efforts to carry out his treatment!) is of greater benefit than the reaction to equally prolonged arterial constriction, (3) that reactive hyperemia is produced by severing large vessels (Leriche makes a point of this), and (4) that an accumulation of metabolites in the tissues is responsible for the local dilatation of the finer vessels (a contention of T Lewis and others)

The most ambitious attempts to secure peripheral vasodilatation by making use of venous hyperemia have taken the form of apparatus designed to secure suction upon a limb followed by positive pressure, the two alternating in a rhythmic manner The limb, being placed in an air tight chamber, is subjected first to negative pressure, during which phase blood is drawn from the capillary bed into the venules and larger veins, after which the limb is emptied of blood by a brief exposure to positive pressure Hermann and Reid maintain that the flow of blood through the smaller arteries and arterioles is thus increased and that the surface temperatures are correspondingly raised By a very similar method, and depending upon Poiseuille's law—the volume per minute of fluid passing through a rigid tube is increased in proportion to the fall in pressure along the tube—Landis and Gibbon undertake to amplify the flow of blood through the peripheral vessels In both cases, suction and pressure are mechanically controlled, a series of cycles being used, but Hermann and Reid use lower pressure for shorter periods, their suction being limited to 80 mm of mercury (about diastolic level) for fifteen seconds and their pressure to 20 mm for two seconds By Landis and Gibbon's more intense and longer periods there may well be introduced an element of reaction to anoxemia lacking in Hermann and Reid's system However, both

gives the impression of being a passive rather than an active process. Present day literature is so full of explanations of the beneficial effect of this or that method of securing vasodilatation as to bewilder the mind, a state of things partly due to the fact that in a growing field each investigator tends to advocate the method which he has developed and with which he is most familiar.

Although for many decades attempts have been made to treat disorders of the limbs by inducing active or passive hyperemia, Cushing's application and release of a tight rubber tube (1902) seems to have been the first successful attempt to secure a reactive vasodilatation in the face of vascular spasm. The idea undoubtedly was derived from the bright and rapid flush which accompanies the release of a tourniquet. The tightened rubber tubing was intended to paralyze the vasomotor nerves by several minutes of complete arterial occlusion. It has since been shown that occlusion of the arterial supply to a limb, even for as short a time as a few seconds, is followed, on release, by a vasodilatation which corresponds to the oxygen want created by the stoppage. That the large as well as the small vessels react in this way is proved by measuring the total inflow of blood into such a limb, and the event can be counted upon, provided the arteries are elastic enough to respond. Its exact cause is not entirely clear but may properly be regarded as a reaction closely related to that called forth by active muscular exercise and by local injury. Whether the stoppage which is succeeded by such a reactive hyperemia is capable of harm, especially when applied to a whole limb, is another matter, but it must be supposed that unless it permanently widens the area of the vascular bed, the increased flow it brings merely balances the momentary damage done by the occlusion.

*Venous pressure*, applied for a prolonged or short interval, is capable of inducing, less violently, a similar reaction. Although the application of this principle is associated with the name of Bier, apparatus for securing venous hyperemia by suction is perhaps a hundred years older, as Hermann relates

raised, is perhaps able to carry fluids from the capillaries to tissues into which they would not otherwise be able to penetrate. In normal persons, at any rate, heightened venous pressure soon increases the flow of lymph.

The final effect of increased venous pressure, at a therapeutic (subdiastolic) level, is to lower oxygen tension in the tissues and thereby create an oxygen want, a want which must be satisfied, upon releasing the cuff, by a reactive hyperemia. It will be realized, then, that venous pressure, in addition to doing what arterial stoppage does, namely, creating in the tissues an oxygen want, raises the pressure in the capillaries while the venous hyperemia is going on and so confers whatever benefits may be received from that act.

There are yet other means of securing reactive hyperemia. The more complicated and forceful methods of doing so by vascular exercise have hitherto been described. The very simplest one is of course the Buerger Allen system of vascular exercises, which consist simply in elevating, lowering, then actually exercising, and finally leveling and warming the limb. An active hyperemia can also be secured by the fever due to the injection of foreign protein, and there are more over certain vasodilating drugs. All such have or, at least, have had their place in treatment. This will be discussed in a later section.

### TESTING THE ARTERIAL SUPPLY TO A LIMB

Should suspicion arise that the blood supply to a limb is deficient, it is first necessary to discover whether a deficiency actually exists and, next, whether the deficiency is due to an organic narrowing of the arteries or to some temporary sort of contraction, that is, vascular spasm. Naturally, investigations in both directions will be expanded by the ambitious. Is the organic deficiency confined to one limb? Is there arterial narrowing throughout the limb, or is some particular vessel obstructed? Is vascular spasm confined to the large vessels, or is it peripheral, or general? And if the circulation is so deficient that a part of the limb must be sacrificed, how shall a

seem to induce in the smaller arteries and arterioles an increased flow of blood

Reactive hyperemia to pure venous compression is vouched for by the careful physiological observations of T. Lewis and Grant (1925). More recently the clinical studies of Collens and Wilensky (who have invented an apparatus for producing intermittent venous compression) and of De Takats, Hick and Coulter (who have measured the reactive hyperemia with the aid of an oscillometer) have placed intermittent venous hyperemia upon the surgical map. As a result, it may confidently be held that raising the venous pressure, as by a broad blood pressure cuff applied to the thigh, to a height just below that of diastolic arterial pressure, say 40–80 mm. of mercury, will cause, on release, a reactive vasodilatation whose intensity is nearly proportional to the duration of the venous pressure, though actually a duration of not more than two minutes produces the best results. It appears, moreover, that an interval of several minutes should elapse before compression is again applied and that a series of cycles should not last over thirty minutes (possibility of refractory phase). The reaction to venous hyperemia takes place best in a warm atmosphere.

For the benefit of those who are interested in the physiological explanation of this phenomenon, it appears that during the application of the pressure, the volume of the extremity increases for some twenty seconds. In this first phase, the vessels are distended and capillary pressure rises to its limit, that is, to 50–60 mm. of mercury (according to Landis, the average capillary pressure is 32 mm. of mercury in the arterial capillary limb and 12 mm. of mercury in the venous limb). At higher venous pressures, that is, above the diastolic level, petechial hemorrhages occur and doubtless red cells escape from the capillaries into the tissues, appearing in the increased lymph stream. The second phase of venous hyperemia is due to stretching of the vascular bed and tissue edema, for the filtration pressure is then increased over the osmotic pressure in the blood. Thus in arteriosclerotics, whose hydrostatic pressure is often low, the filtration pressure, thus artificially

12° F and soon the normal hand or foot responds with a rise to its natural limit, to 93°-94° F (34.5° C), a range of 23°-25° F

In examining the limb, the observer will use the back of his fingers, having already applied them to his own neck to test their warmth, and will compare the patient's cutaneous temperature with his own. If in a well warmed room the patient's skin feels warm to him, the circulation in the tested limb may be either normal or deficient. But if in a cool room the patient's foot or hand feels cold, patient and observer having been equally exposed, the patient's circulation is almost certainly deficient. Thus the simplest test is to expose the hitherto warmly covered extremities for ten to fifteen minutes to a room temperature of 70° F or lower. If, then, the hands or feet, as the case may be, feel cold to the examiner's touch, the circulation in the patient's limbs is presumed to be insufficient. Even more striking will appear any difference in surface temperature between two symmetrically placed limbs. To take an example. An elderly individual complains of feeling a numb sort of cramp in the left calf on walking two blocks. His feet become cold in winter. He must warm them at night before he can fall asleep. He is often awakened by cramps. If he uncovers his feet and legs in a cool room—after they have become thoroughly warmed in bed—leaving them exposed for some ten minutes, both toes and feet feel cool to the touch. At the end of fifteen minutes the left foot is distinctly colder than the right. As the hand is passed down the leg, from knee to toes, the coolness is felt to increase from above downwards, but there is no abrupt change as the foot is approached (absence of vasomotor spasm or thrombosis of a large artery) and the foot is dry (absence of associated sudomotor excitation as a sign of sympathetic vasoconstriction). A case such as this will usually present a feeble or absent pulsation in either the dorsalis pedis or posterior tibial artery (or both) of the affected limb or limbs. The color tests usually associated with such a state will presently be described. However, there is little doubt that this is an arteriosclerotic deficiency of



safe level for amputation be determined? To carry the story of investigation further would lead to a differential diagnosis between the various known states of vascular disease. It is only intended here to describe the various known means of studying the vascular capacity of the limbs. Some of these means are simple, to be used on ambulatory patients and at the bedside. Others are complicated and call for elaborate and often expensive apparatus. For anyone willing and able to use his eyes, his fingers and his commonsense, the simpler methods afford most of the diagnostic tests he needs. The more complicated methods correct and amplify the simpler ones, explain obscure signs, and furnish the accurate data needed to reveal the exact results of treatment.

#### AMBULATORY AND BEDSIDE TESTS

**The Temperature of the Skin**—This indicates very responsively the rate of the blood flow through the limb. That is, the more rapid the flow, the greater the heat lost from the surface and the warmer to the touch the skin actually feels. There is a certain normal background. Exposed to a surrounding temperature of about 68° F (20° C) or below, normal hands and feet tend to be cool, to feel cool to the observer's touch, having an actual temperature, as determined by special apparatus, of somewhere about 70° F, rarely much higher and never more than a few degrees lower. In warm surroundings, say at 78°–80° F (about 26° C) or higher, the skin of the hands and feet tends to show a temperature of 90°–94° F (about 32°–33° C). Actually the tips of the fingers and toes offer the most striking changes of this sort, because of the very sensitive arrangement for vasoconstriction and vasodilatation which the digits possess. However, the thin skin of the dorsum of the hands and feet does very well as a test surface. In the influence exerted by the room temperature upon the surface temperature there is a sort of critical level or rather hill. Drop the room temperature below 70° F and local circulation is slowed, the skin temperature falling nearly to that of the air about it, but raise the room temperature only perhaps 10°–

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### AMBULATORY AND BEDSIDE TESTS

**The Temperature of the Skin**—This indicates very responsively the rate of the blood flow through the limb. That is, the more rapid the flow, the greater the heat lost from the surface and the warmer to the touch the skin actually feels. There is a certain normal background. Exposed to a surrounding temperature of about  $68^{\circ}\text{F}$  ( $20^{\circ}\text{C}$ ) or below, normal hands and feet tend to be cool, to feel cool to the observer's touch, having an actual temperature, as determined by special apparatus, of somewhere about  $70^{\circ}\text{F}$ , rarely much higher and never more than a few degrees lower. In warm surroundings, say at  $78^{\circ}$ – $80^{\circ}\text{F}$  (about  $26^{\circ}\text{C}$ ) or higher, the skin of the hands and feet tends to show a temperature of  $90^{\circ}$ – $94^{\circ}\text{F}$  (about  $32^{\circ}$ – $33^{\circ}\text{C}$ ). Actually the tips of the fingers and toes offer the most striking changes of this sort, because of the very sensitive arrangement for vasoconstriction and vasodilatation which the digits possess. However, the thin skin of the dorsum of the hands and feet does very well as a test surface. In the influence exerted by the room temperature upon the surface temperature there is a sort of critical level or rather hill. Drop the room temperature below  $70^{\circ}\text{F}$  and local circulation is slowed, the skin temperature falling nearly to that of the air about it, but raise the room temperature only perhaps  $10^{\circ}$ –

*Warm, Deep Red Skin*—Such a skin is warm because it is inflamed or because vasodilatation has been brought about by some artificial means, such as reactive hyperemia or drugs (nitrites)

*Warm, Deeply Cyanosed Skin*—Unless the hemoglobin is altered (as by such a drug as sulfanilamide) such a skin is cyanosed because the circulation is delayed and warm because of external heating

*Cold, Pale Skin*—Such a skin is cold and pale because the blood flow is restricted. A slow or absent circulation gives a cyanotic tint

*Cold, Deeply Cyanosed Skin*—Such a skin is cold because the circulation is deficient or absent, and cyanosed because the circulation has been slow for so long a time that the blood has given up its oxygen. This is not inconsistent with a low grade inflammation

*Cold, Deep Red Skin*—If the skin is very cold, the blood will not give up its oxygen and since the small surface vessels are injured and therefore dilated, the skin is deep red. Blood flow, however, is very slow

*Momentary Pressure upon the Skin*—This test, often used, is only moderately informative and may indeed be deceptive. If, for instance, the great toe is deep red, the expression of blood from the skin by pressure will be followed by a very rapid return of color. This does not indicate, if the toe is cold, a vigorous circulation, but merely the pressure at which blood is standing in the near by vessels. In this case, a cold, red great toe would be the seat of a very slow circulation. Indeed the circulation might have ceased, yet the blush would return after pressure, as in any dependent part

Again if a cold, already pale area is made paler by pressure, the return of color must be slow because the finer vessels in a cold skin are contracted. But this does not mean that the circulation is deficient

The test is most valuable when applied to an elevated extremity. In the presence of a vigorous circulation, blanching due to pressure upon an elevated part is promptly followed

moderate severity but affecting the left leg more than the right

A variation upon this test of temperature can be made if the room is warm (80° F or over) The legs from the knee down are immersed in cool water, that is, considerably below the room temperature They are then dried and left exposed as before In that case, the feet and toes inadequately supplied with blood will be slow to become warm, or one leg will lag behind the other Normally, the maximum rise should be secured in fifteen minutes If, on the other hand, the room is cold, that is, well below 70° F, it may be best to start by immersing the feet in hot water at body temperature Thoroughly warmed in this way, dried and exposed, their rate of cooling can easily be discovered

**Test by Color**—Since color is given to the surface by the minute vessels of the skin itself, it is a less reliable test of the circulation in the limb as a whole than is the temperature However, a white skin, that is, an excessively pale one, indicates a restricted circulation A bluish skin indicates a slow circulation, one which may or may not be abundant Redness shows that plenty of blood is present, owing to inflammation or heated surroundings, which has not yet lost its oxygen The tint, in fact, as Lewis points out, must be interpreted in the light of the skin temperature For example, the cool, arteriosclerotic foot described in the previous section is pale Coldness and pallor indicate a diminished circulation, probably *not* particularly slowed But if this same foot were cold, pale, and bluish, the circulation must necessarily be not only diminished but much delayed (for since cold blood is slow to give up its oxygen it must have been long delayed to become blue and not red) As an aid to an understanding of color and surface temperature, Sir Thomas Lewis's interpretations, somewhat amplified, are here reproduced.

**Warm, Pale (Pink) Skin**—Such a skin is warm because for some time blood has flowed rapidly through it and pale pink because the skin is well nourished, causing vasomotor tonus (moderate vasoconstriction) to be normally present

toes, the tips of which may not show color for half a minute or longer. Such a test does not relax vasospasm, for merely raising and lowering the leg fails to call forth the same degree of reactive hyperemia which sets in after the circulation to the whole limb has actually been shut off. Thus it does not differentiate organic from spasmodic obstruction. Other observations, however, may already have settled this point. If not, a somewhat more elaborate test, described by Lewis, may be used.

*Heating, Elevation and Depression to Secure Reactive Hyperemia*—Though this test requires no complicated apparatus, its various steps call for strict attention to detail. The room in which the test is made should be warm. Relaxation of the blood vessels of the limb to be tested must first be secured by warming it for perhaps ten to fifteen minutes. The foot bath, maintained at blood heat, is best for this purpose. The limb is then dried and raised somewhat above the level of the body until the skin becomes pale. Thus the smaller vessels are relaxed and empty. Using a blood pressure cuff, the arterial supply to the limb is now shut off for five to ten minutes by maintaining a pressure exceeding the systolic pressure. (There seems no good reason for keeping the limb in the water bath during the application of the pressure, as Lewis directs, provided the room is warm, 78° F. or over.) On release of the pressure, the relaxed vessels are rapidly filled so that the skin becomes bright pink, to the very tips of the toes, in two to five seconds.

Should the blood vessels be diseased, the toes may not turn pink for half a minute or even longer. Or some toes will color long before others, giving information as to the areas most seriously deficient. The flush dies out most rapidly in the parts in which it first appears and lasts longest in the regions most slowly colored. An organic deficiency is clearly revealed by this test and can usually be distinguished from vascular spasm. In the latter case, the extremity, cool, damp, and tending to be cyanotic, beforehand, is made to flush like a normal limb. Or if spasm is superposed upon some degree of organic con-

by a return to a normal if only faintly pink color. But in the presence of a feeble circulation a blanched elevated part regains its natural color only after many seconds. The experienced observer is able to harmonize the different periods of delay in the return of color to a pressure spot (the limb being slightly elevated) with other tests.

#### SIMPLE TESTS OF VASCULAR OBSTRUCTION AND OF CAPACITY FOR VASODILATATION

Though the more elaborate tests are required to ascertain the exact state of the arterial supply to a limb, one or two simple ones will give a surprising amount of information.

*Elevation and Depression*—The leg having been exposed in a warm room for ten to fifteen minutes to obviate any accidental vasomotor constriction, is examined for surface temperature and color, the patient lying supine. It should now be raised to an angle of  $30^{\circ}$ – $45^{\circ}$  with the body and held in that position for a period of perhaps two minutes. The color of the foot is noted, that is, whether it retains a healthy pallor or takes on a cadaveric, yellowish white shade. In the latter case, the arterial supply is deficient, possibly, if the blood pressure is low, only relatively deficient. In this position also the characteristic cramp of intermittent limp is easily brought out automatically or by exercising the foot, and, as Samuels points out, such exercise emphasizes any deviation from the normal pink color of the sole, especially any difference between the two feet.

On lowering the leg, the patient sits against the edge of the bed, the legs resting on the floor. If the circulation is normal, a flush appears in a few seconds, first at the ankle, and quickly spreads to the feet and toes which take on a pink blush without blueness. The whole process takes perhaps five to ten seconds or less. If, however, the arterial supply is imperfect, there may be a pause of ten or more seconds before the flush appears on the foot at all, after which it usually progresses in a deliberate and perhaps irregular way, slowly reaching the

vasomotor system now in use. The next step, still a clumsy one, was the trial of various general anesthetics for a similar purpose. But, though any anesthetic capable of carrying a patient into the stage of full relaxation (and even some gases which hardly go so far) paralyzes at the same time the sympathetic nerves, such a method is hardly adapted to general use.

The solution of the problem was found in regional anesthesia by procaine. Spinal anesthesia confers a complete sympathetic paralysis upon the region anesthetized, which in the great majority of cases is the lower half of the body. It seemed at first to serve ideally for a study of the circulation in the legs, and still is the most definitely positive method of securing full vasodilatation of the lower limbs. However, when White proved that blocking the sympathetic supply to one limb could be accomplished by paravertebral injection of the various sympathetic ganglia or by anesthetizing a great nerve trunk, he opened the way for comparing the state of two symmetrical limbs—a most desirable event. It became clear also that single peripheral nerves, the sole source of sympathetic impulses to certain cutaneous fields, could be blocked with procaine, a matter explained by the much earlier anatomical observations of Kramer and Todd, Potts and others. (See Figure 2.)

The story would be incomplete without reference to the vasodilatation made possible, principally through the observations of Lewis, that heating the blood of one part of the body sets off a physiological reaction (vasodilatation) which warms all the limbs. Collier and Maddock found that heating the body raises the surface temperatures of the exposed extremities and that vascular deficiencies cause this mechanism to fail. Gibbon and Landis went a step farther and secured vasodilatation in the feet by immersing the arms in hot water.

As for drugs, a good drink of alcohol gives a very efficient dilatation, and recently Beck and De Takats have found that the administration of a standard dose of sodium nitrate offers



striction, the reactive hyperemia will be rapid, but the flushing will fall short of completeness. All such fine points will only be settled by the more elaborate tests which make use of the surface temperature, or the actual blood flow through the extremity, as measures of the actual degree of vasodilatation.

### TESTS REQUIRING SPECIAL APPARATUS

To be accurate, tests for vasodilatation should record the actual surface temperature and insure in the field to be studied the most complete vasodilatation possible. Practically speaking, there is no such thing as an accurate record of surface temperature—the radiation of heat being at the mercy of too many influences—but the use of the thermocouple is decidedly the most satisfactory method. It is in securing vasodilatation that the variety of methods becomes confusing. A brief review of these will explain why certain ones have survived to become standard.

The test by the introduction of foreign protein, particularly typhoid vaccine, was introduced by Brown (1926). Such a substance, after causing a preliminary vasoconstriction (chill), calls forth a high fever of a few hours' duration and a universal relaxation of the blood vessels. The vasodilatation and rise of temperature, according to the principles already laid down, are most marked in the digits. And by balancing this rise in the tips of the extremities against that of the body temperature (mouth or rectal) a ratio or vasomotor index can be determined. In simple language, the greater the rise of temperature upon the digits in proportion to the rise of temperature in the body at large, the more completely vasoconstriction has been relaxed and vasodilatation secured. Moreover, even if no physiologic or pathologic vasoconstriction has been present, the test reveals whatever capacity sclerosed vessels have for relaxation. Unfortunately, it is disagreeable to the patient, on account of the chills and malaise, and is not without danger. It is not only variable, but both surface and body temperature are unfixed. However, it opened the way to the more reliable and convenient methods of examining the peripheral

that he feels no sense of coldness or of heat. When the skin temperatures are running at a constant level, which will usually be somewhere below  $80^{\circ}\text{F}$  ( $27^{\circ}\text{C}$ ) the patient should be turned upon one side for the spinal anesthesia. In this position the contact wires will not be disturbed. He should be uncovered as little as possible.

For most adults, procaine crystals to the amount of 150 mgm in four ccm of spinal fluid will give a satisfactory anesthesia up to a point well above the umbilicus. The needle should be inserted into the second or third lumbar interspace. The injection is made with the patient's body horizontal, after which he is returned to the supine position. The foot of the bed may then be raised four inches, though this is hardly necessary. In any case, the head should not be raised on a pillow during the anesthesia (danger to medullary centers). If the blood vessels of the legs are capable of vasodilatation, the rise of temperature will be maximal, that is, to  $91^{\circ}\text{--}95^{\circ}\text{F}$  ( $33^{\circ}\text{--}35^{\circ}\text{C}$ ). If the vessels are in a state of spasm, such spasm will be relaxed and the same high temperatures will be reached. But if organic constriction is present—arteriosclerosis or thrombo angitis obliterans—the rise will be altogether absent or of only a few degrees. Expect the rise in five to ten minutes except in cases of obstinate vasospasm when it may be delayed for fifteen to twenty minutes. Any anesthesia which extends upward to the level of the clavicles will cause full vasodilatation of the arms.

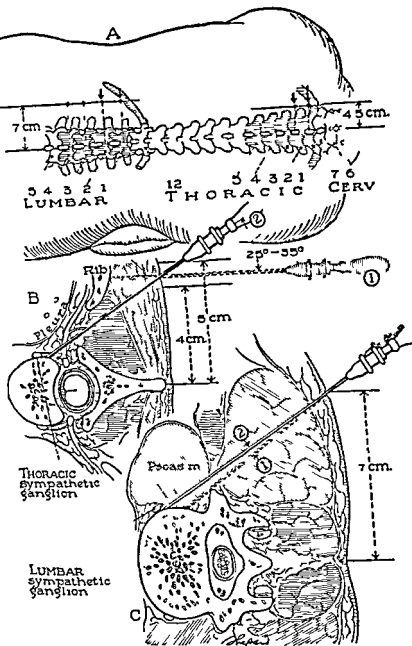
**Paravertebral Anesthesia**—For practical purposes, this is the only method of securing paralysis of vasoconstriction for one entire limb. If successful, the degree of vasodilatation in that limb is, theoretically at least, as complete as is the case with spinal anesthesia. Those who desire the most detailed description should consult the writings of White, who first advocated the procedure, and of Flothow, who has gone on logically to develop it into a means of securing a prolonged vasodilatation by the injection of alcohol. The accompanying illustration will bear out the statement that precise directions are of little value. The operator must have the ability to visualize the spinal column, its transverse processes and the ribs, and

a safe and reliable means of studying the capacity of the peripheral arteries for relaxation

All such procedures must be judged by the completeness with which they abolish vascular spasm. In general, spinal anesthesia for the lower limbs is most reliable. Heating the body, or a part of it, is subject to the objection that any one vascular spasm may be so severe as to fail to be influenced. Paravertebral nerve block would be ideal if it were not a little too dependent upon the skill of the operator. And peripheral nerve block only affects the peripheral vessels, that is, vasomotor control of the great arteries central to the block is unaffected. Nevertheless, both the local application of heat and the various regional anesthetics are extraordinarily informative—all the more so when means of taking surface temperatures are at hand.

Modern means of recording surface temperature were first developed by Benedict and his associates. Now various instruments based on the thermocouple are available. None is strictly accurate. The most one can say of any is that with a moderate amount of care, the changes in the temperature of *any one part can grossly be recorded and comparisons made* between two symmetrical limbs. For the most accurate work a room, free from drafts, whose temperature can be controlled is required. Self recording apparatus is very convenient. Doubtless the technique in this field will continue to develop.

**Spinal Anesthesia**—To take full advantage of the complete vasoconstriction secured in the anesthetic field by spinal anesthesia, cutaneous temperatures should first be taken from at least four points upon the feet, by wire loops attached with light silk about the toe or instep, during perhaps an hour's observation in a room neither hot nor cold but kept at a constant neutral temperature—anywhere between 70° and 75° F or 21°-24° C. The four points should be the dorsal surface of each great toe just behind the nail and the instep of each foot (The thickness of the skin of the sole makes this surface unreliable.) While the records are being made, the thighs and body of the patient should be covered with light blankets so



**FIGURE 3 SYMPATHETIC BLOCK FOR THE ARM AND LEG** For the arm, the points indicated by arrows are opposite the spinous processes, for the leg, opposite the interspaces. In making the injection of procaine it is usually sufficient to inject at T, 1 for the arm and at L, 2 for the leg

to know where the point of his needle is going. Above all he must have the disposition suited to local anesthesia, the gentleness, deliberation and power of assurance without which injections of procaine are a trial to the patient, in some cases, an agony.

*Upper Thoracic Block (the Arm)*—The sympathetic ganglia at this level lie about opposite the middle portion of the vertebral body, that is, from front to back, and very close to the pleura. To reach them safely the needle must penetrate between two ribs, and, about an inch (two and a half cm) deeper, that is, in the direction of the front of the vertebral body, but the body. If the injection is made with the needle in this position the solution must bathe the sympathetic chain.

The ganglia to be reached are the first and second thoracic. Injection of the first usually paralyzes the whole sympathetic supply for the arm, but injection of the second, as well, is occasionally required. Wheals are made four to five cm lateral to the upper two thoracic spines and through these, using a longer needle, procaine is injected down to the sensitive surface of the underlying ribs. The eleven cm needle (with a filler, and without an attached syringe) is now thrust in directly until it meets the posterior angle of the rib. It is then withdrawn, directed toward the midline at an angle of about  $25^{\circ}$ – $35^{\circ}$  until, at a depth of about an inch (two and a half cm) frontal to the rib, it meets the side of the vertebra. Here the operator's sense of position must guide him. If the patient is large and heavily muscled he will have started his insertion fully two inches (five cm) lateral to the spine and will have farther to go to reach the vertebral body. In any case, if he feels he is striking the body too far posteriorly he must alter the direction of his needle and push for a contact a little farther forward.

On inserting the first needle, he will do well to attach an empty syringe and suck to see if he can draw out either air or blood. If he secures air, he must have punctured pleura and lung and must reinsert the needle, hugging the vertebral column closer. If blood, he must withdraw and reinsert the needle

cem. of one per cent procaine are injected. Actually, such an injection opposite the second lumbar interspace will usually block the whole lumbar sympathetic chain (since a liberal amount of the procaine solution passes freely up and down the retroperitoneal cleavage plane) but the third interspace is often injected for additional assurance of completeness. An injection of one side will occasionally pass across the mid line, affecting the opposite leg.

It should be remembered that any anesthesia which may result will pass toward the pubes and that the patient can experience no numbness in the leg or foot. Thus the sensation of warmth, and in case pain is abolished, of comfort, is very readily noticed. A comparison of the state of the two legs is of course a prime object of the test.

*Peripheral Nerve Block*—The vasodilatation secured by an injection of procaine about a great peripheral nerve such as the posterior tibial, median, or ulnar, falls short, as Morton and Scott have proved, of being complete, that is, after a rise of surface temperature to a constant level has been obtained by the peripheral injection, spinal anesthesia will evoke a still further rise. The failure of the peripheral block to secure a maximal vasodilatation is presumably due to the normal, high vasomotor tone in the great arteries central to the block, a tone untouched by peripheral nerve block but abolished by spinal (and probably paravertebral) anesthesia. To bring out this point, one of Morton and Scott's charts, somewhat simplified, is herewith reproduced. It will be noted that spinal anesthesia raises the surface temperature of both great toes from a level of about  $71^{\circ}$ – $72^{\circ}$  F ( $22^{\circ}$  C) to at least  $89^{\circ}$  F ( $31.5^{\circ}$  C) and as a rule nearly to  $93^{\circ}$  F ( $34^{\circ}$  C), but posterior tibial block only raises the temperature of the great toe to between  $87^{\circ}$  F ( $30.5^{\circ}$  C) and  $89^{\circ}$  F ( $31.5^{\circ}$  C). In fact Morton and Scott go so far as to establish a "normal vasodilatation level" for both spinal (or general) anesthesia and for peripheral block anesthesia. These levels are subject to correction for variation in the room temperature ( $0.54^{\circ}$  F or  $0.3^{\circ}$  C to be added for every degree of room temperature above  $68^{\circ}$  F or

a little higher or lower After inserting the needle for the first thoracic (stellate) ganglion, making suction and injecting a few drops of procaine to see if the patient tastes the fluid or coughs, five to twenty cc. can safely be introduced

A successful block of the stellate ganglion will cause contraction of the pupil as well as dilatation of the peripheral vessels of the limb (if these are dilatable) A subjective sense of warmth will be felt in the hand, whose surface will become dry and warm to the touch The cutaneous temperatures should reach a peak in a few minutes By inserting the needle from above the first rib—in a direction more caudad than usual—the risk of piercing the pleura and lung is less and the pupillary reaction is an even surer test of success

*Lumbar Block (the Leg)*—For the lumbar injection the technique must be varied slightly on account of the greater size of the vertebral body and of the forward position of the sympathetic chain The wheel is placed opposite the interspace instead of the spinous process and at a distance of about two and a half to three inches (six to seven cm.) from the midline To fall opposite the second lumbar interspace it must be made very close to the twelfth rib Through the wheel opposite the second lumbar interspace, the muscular aponeurosis, which is sensitive, is carefully infiltrated with the one per cent solution The long needle often meets the tip of a transverse process and helps in the estimation of depth, for the point to be reached is rather over an inch (three cm.) anterior to the process and more than three inches (eight cm.) from the surface The needle is thrust in at an angle of about 45° toward the vertebral body, just meeting the body full on its lateral aspect The succeeding adjustment requires some skill The needle is first withdrawn and redirected farther forward striking the body at a still greater depth Then, ideally, it is adjusted so that it glides past its last contact with the body to a depth one fourth to one half an inch (one cm.) greater Here its point lies on the anterior edge of the psoas muscle and just behind the aorta or vena cava, here suction is made to determine whether or not a blood vessel has been injured and here ten to twenty

upper edge of the great trochanter to the posterior superior spine (isiotrochanteric line), a perpendicular is drawn downward upon which a point is selected one and a quarter inches (three cm.) from the first line. Here a needle, four inches (ten cm.) long, is carefully inserted in a direction normal to the surface until it causes paresthesia in the course of the sciatic or meets bone at a depth of two and one fourth to three inches (six to eight cm.) The nerve should be looked for at about a depth of two inches (five cm.) It may be necessary to feel for the nerve which should not be pierced. Ten to twenty cc. of a two per cent solution of procaine may be injected. Anesthesia should appear in ten to twenty minutes. It is doubtful whether sciatic block can be expected to give a maximal vasodilatation.

*Posterior Tibial Block*—This block gives, for normal vessels, an incomplete but constant vasodilatation and rise of temperature to between 87° and 89° F (30.5° to 31.5° C). The nerve is found below the internal malleolus, where it is easily palpated as a hard round cord behind the posterior tibial artery. Having made a wheal over it with a fine needle, fix the nerve with the index finger of the other hand and introduce ten cc. or so of two per cent procaine into the fascial compartment in which the nerve lies. The nerve itself should not be pierced. The skin of the heel, sole, and plantar surface of the toes soon becomes wholly or partly anesthetic and a flush appears. The skin of the sole is so thick that the temperatures are best taken from the plantar surfaces of the outer phalanges of the great and little toes.

*Median Nerve Block*—Though the median can be blocked at the wrist, its large size and tough sheath make its injection rather unsatisfactory as a means of securing a peripheral vasodilatation. The guide is the tendon of the palmaris longus muscle. By resisted flexion of the wrist this tendon and that of the flexor carpi radialis (on the radial side of the former) are brought out. The nerve lies between the two. Through a wheal at the level of the radial styloid, just lateral to the tendon of the palmaris longus, the needle is introduced directly through the deep fascia. When the vicinity of the nerve is reached,



20° C ) but for practical purposes are those described above. The usefulness of peripheral block anesthesia lies, of course, in the fact that the temperature level it secures is just as reliable a test of peripheral vasodilatation as spinal or general anesthesia. The defect of the method really lies in the failure of procaine in some cases fully to anesthetize a great nerve such as the sciatic, posterior tibial, median, or ulnar.

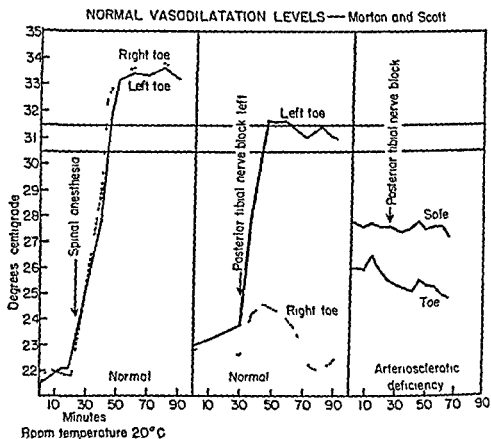


FIGURE 4. NORMAL VASODILATATION LEVELS FOR SPINAL AND PERIPHERAL SYMPATHETIC BLOCK (after the Charts of Morton and Scott)

*The Sciatic Nerve*—The nerve is reached as it comes out through the great sacrosacral foramen, just below the piriformis muscle. Labat's technique is the following:

The patient lies on the side opposite to the one to be injected in the Sims position, that is, both legs drawn up, the knee corresponding to the nerve to be injected a little overlapping the other. From the middle of a line traced from the

controlled room, especially when means of continuously recording surface temperature are available, affords perhaps the most luxurious and foolproof means of studying the vasomotor reactions in the extremities. The temperature of the digits having been recorded in a cool room, the body can be heated to secure the maximal vasodilatation.

### Tests by the Use of Drugs

*Alcohol*—A stiff drink of whiskey or any strong liquor is, except for the corruption of the individual, an admirably simple and practical method of testing the ability of the vascular bed to dilate. Unless a very accurate check on such vasodilatation is needed, the oscillogram will give a sufficiently good idea of the patient's response. His own sensations will usually inform him that the dose has been sufficient and that all the dilatation possible under the circumstances has been secured. The test is of some value in estimating very roughly, in the presence of an intermittent limp, the elasticity of the circulation and the possibility of improving the arterial supply to a limb by (permanent) sympathetic block. Some individuals, for instance, will be able to walk twice as far, without limping, after the dose of alcohol as before. On the whole, the method is rather on the rough and ready side—for a snap-diagnosis.

The use of sodium nitrate, intravenously, in a dose of one ccm. of a four per cent solution has recently been advised by Beck and De Takats. The oscillogram is used to record the amount of vascular relaxation. The effect appears in ten to fifteen minutes and lasts perhaps an hour. In this dosage, the drug apparently is not dangerous.

### Arteriography

By exposing a limb to the X ray during the injection of certain solutions \* opaque to the X ray, much information has been secured as to the exact state of the arteries in vascular

\*Thorium dioxide or 'Thorotrast' has been much used. As a radioactive substance objections to it have been raised. It certainly is not excreted, is probably retained in the liver and may hypothetically and after many years cause

paresthesia in the median field is apt to be noticed Ten to twenty ccm of a two per cent solution of procaine is then introduced If no paresthesia appears, the needle can be directed a little farther radialward Massage aids in bringing on the anesthesia

*Ulnar Nerve Block* —This is best made at the elbow where the nerve is easily palpable posterior to the internal epicondyle of the humerus Through a wheal over the nerve a fine needle is introduced into its vicinity (not actually piercing it) A two per cent solution of procaine is liberally injected The area supplied by the ulnar nerve becomes pink and dry, giving the patient, as a rule, a sensation of warmth and numbness, the anesthesia being incomplete but vasodilatation satisfactory

### Exposure to High and Low Temperatures

The most practical means of using heat to secure vasodilatation is to immerse the hands and arms in hot water The usual arm basins serve the purpose The water should have a temperature up to 110° F (43° C), that is, a heat just bearable If an accurate account of the rise of surface temperature is to be kept, the usual preliminary control observations should be made at a room temperature of about 70°–74° F An immersion of fifteen minutes will usually cause a rise of temperature in the feet, and, except in the face of an obstinate vasoconstriction, the rise will in most cases be maximal Obviously the test can be reversed Beginning with warm surroundings, and a high level of surface temperature for the feet, the arms can be immersed in cold water But here the fall will merely record the promptness of the resulting vasoconstriction There is no ideal end point for the observation On the whole, the responses to immersion of the arms in hot water can not be given full authority Lack of response in the form of a rise in the surface temperature of the feet can not be regarded as proof that no possibility of vasodilatation exists To test the arms, the feet and legs can be immersed in hot water, a rather awkward procedure and not entirely satisfactory

A jacket, electrically heated, for use in a temperature

controlled room, especially when means of continuously recording surface temperature are available, affords perhaps the most luxurious and foolproof means of studying the vasomotor reactions in the extremities. The temperature of the digits having been recorded in a cool room, the body can be heated to secure the maximal vasodilatation.

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Thorium dioxide or Thorotrast has been much used. As a radioactive substance objections to it have been raised. It certainly is not excreted, is probably retained in the liver and may hypothetically and after many years cause

disease of the limbs. The variations in the caliber of arteriosclerotic vessels, the situation of an obstruction in the form of an embolus or thrombus, and above all the nature of the collateral circulation in serious arterial deficiencies have been observed. Yet it cannot be said that arteriography is of any vital diagnostic aid, and perhaps so far it has been most successful in confirming the impressions acquired by simpler tests. It should probably be practiced only by the most expert, not entirely because of the danger inherent in the procedure but because perfect technique alone justifies an exact interpretation of a picture in a critical case.

There are minor variations in the method of introducing the opaque material, depending upon whether the act is performed by a "team" or by an individual. There is, however, a general agreement upon the following points:

1. To visualize the vessels of the forearm and hand the injection is made into the brachial artery just above the elbow. At this level five ccm. of the solution are sufficient and more than ten ccm. are never required.

2. The vessels of the forearm and hand are best visualized in the antero-posterior position.

3. To visualize the vessels of the lower thigh, the knee and the leg, the injection is made into the femoral artery within the femoral triangle (below the giving off of the profunda). The best view is secured when the exposure is almost lateral, the leg externally rotated and the knee placed against the plate. Twenty ccm. of the solution are required.

4. To visualize primarily the vessels of the leg, ankle and foot, the injection is made into the femoral artery in Hunter's canal. The exposure should be made in an antero-posterior direction. Not more than twelve ccm. of the solution are required.

The method used by Veal and McFetridge for injection into the femoral artery is the following. The skin over the site of

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serious damage. Such damage, however, has not yet been proved inevitable in the dosage used.

Iodine solutions, such as are used for the purpose of making intravenous pyelograms, give less vivid shadows but are without danger to the individual. Some of them, however, cause pain when injected. Such solutions as "Diodrast" seem reasonably satisfactory. Doubtless new and superior ones will be invented. Thus far, all the solutions used cause some degree of vascular spasm.

injection is anesthetized with one per cent procaine. The artery is punctured with a number eighteen needle attached to a syringe filled with the solution. As soon as the puncture is accomplished (bright red blood enters the syringe in spurts) pressure is made with the thumb just proximal to the site of the puncture, stopping the spurt of blood into the syringe. At once, the injection is made, the thumb continuing to compress the artery. When all the solution has entered the vessel, the pressure of the thumb is released, allowing the distal tree to be filled. Three to six seconds later, depending upon the position of the puncture and the length of the limb, the exposure is made.

For the brachial injection (at the elbow) a Wassermann needle is used, the syringe containing the solution being attached. A local anesthetic is hardly required. Immediately upon entry into the vessel, digital pressure is made just proximal to the puncture. Pressure is released and the exposure is made as soon as the injection is concluded.

The method of injection used by Allen and Camp is somewhat different. For the brachial injection, a blood pressure cuff is first placed upon the upper arm as near as possible to the shoulder. The artery is punctured with the needle attached to the syringe containing the solution. When blood spurts back through the needle, the cuff is inflated to systolic pressure, shutting off the circulation. The solution is then injected, the needle withdrawn and an exposure is made at once. But now the cuff is deflated to the diastolic level for two to four pulse beats. Upon its re-inflation, to shut off the flow, a second plate is taken. The procedure can be repeated for the taking of a third plate. Obviously this procedure requires nice team work.

For the femoral injection, the needle is introduced, the artery shut off proximally by pressure with the fingers, and the injection made. On withdrawing the needle, the artery is released for a few beats, an exposure made, and the vessel again compressed. As in the case of the arm, the release, exposure, and compression can several times be repeated.

### Blood Flow as a Test of the Arterial Circulation

The flow of blood through the foot has recently been used by Kunkel and Stead as a measure of the efficiency of the circulation in the lower extremity. In their publication, they state that they have modified for this purpose the apparatus of Hewlett and Van Zwaluwenburg as well as that of Freeman, intended to measure the flow of blood in the hand. The apparatus is a plethysmograph (water bath) in which the foot is first accustomed for half an hour to a temperature of 33° C. "When the venous outflow is occluded by a 'collecting pressure' lower than the diastolic pressure, the rate of the initial increase in the foot volume is a measure of the amount of blood flowing to the foot." The result is expressed in so many ccm per minute per 100 ccm of foot.

Expressed as above, the average blood flow of normal subjects was found to be 17.1 ccm (the highest 25.9, the lowest 11.1 ccm). This is about one half the flow estimated in a similar way for the hand. The investigators found that a fifty per cent reduction in the flow of arteriosclerotics and sufferers from thromboangitis obliterans was not associated with symptoms or external signs. Beyond this point, evidences of arterial deficiency were usually noticed. A rather interesting finding was the discovery that an intermittent limp might be present though the flow was seemingly sufficient to have obviated it. That is, the muscles of the calf might be ill supplied, though the foot received a good flow of blood.

The test is interesting as a check on others but seems, at the moment, to hold no advantage over less cumbersome methods. However, the apparatus, when perfected, should be far less expensive than an oscillograph or the thermocouple galvanometer machine for recording surface temperatures.

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## CHAPTER II

### ARTERIOSCLEROTIC DEFICIENCY AND THROMBOSIS

THE pathological background of arteriosclerotic peripheral vascular disease is narrowing of the vascular channel due to a chronic, progressive thickening of the intima. This is not an orderly or an evenly distributed process. There is intimal proliferation, generally most marked on one side or another of a vessel, so that a semilunar thickening, as seen in cross section, is built out into the stream. Hyperplasia of the elastic tissue in such areas is followed by atheroma and calcareous deposits. By such means the lumen is narrowed and elasticity is lost. Just what makes this process vary so much from individual to individual is a mystery. So far as the limbs are concerned, it is always more advanced in the lower than the upper, perhaps because arterial pressures are decidedly higher in the legs than the arms. One can adduce as causes mental and physical strains, infections and other influences over which human beings have little control, but of all adverse factors diabetes seems to be the most powerful. Diabetics suffer at an earlier age than do others from arterial deficiency in the legs, and apparently insulin does not protect against this change.

Arteriosclerotic narrowing and hardening leave a vessel of irregular caliber. Fibrous thickening and deposits of calcium are most marked at bifurcations and points of active bending. Thus the vessels of the groin, the popliteal region and upper calf in particular are most seriously affected. Once encroachment on the lumen has begun, there is a tendency to a deposit of platelets and so to thrombosis. A sudden closure brought on in this way cuts off the arterial stream from a considerable area and causes ill nourishment, if not actual gangrene, in the

field served by the occluded vessel. By contrast, gradual constriction of an artery is accompanied by the development of so effective a collateral circulation that the peripheral parts may remain well nourished. On the one hand, anoxemia is sudden and gangrene follows; on the other, the opening of new channels so nearly keeps pace with contraction of the old that function need never be disturbed. When the development of a collateral circulation barely keeps pace with arteriosclerosis, the stage is set for intermittent claudication and the so called "trophic" disorders of the nails and skin, a state of things which may be prolonged, without any *serious* disability, for many years.

In arteriosclerotic deficiency, the small collateral arteries, like the main vessels, are often irregular in caliber and so erratic in distribution as to permit a more satisfactory blood supply to reach one area than another. The toes, perhaps, receive a sufficient circulation but the muscles or some one group of them does not. Or the muscles are well looked after and some or all of the toes are ill nourished. Naturally the great muscles, which require far more blood in action than at rest, are unable to function normally. The individual finds that after walking several blocks, one leg or the other feels numb or cramped, or even as if stuck with a knife. He rests for a minute or two, finds himself comfortable and steps out again, only to have the same pain return after about the same distance is covered at the same pace. This intermittent limp may become worse or remain fixed at the same point or may improve, according as the collateral circulation is able to respond to the deficiency. But it is important that its nature be recognized, and a misfortune when the patient's disability is attributed to "fallen arches."

In a recent study of arteriography, Veal and McFetridge show how the pathological background can explain the clinical appearances, for example, that a relative lack of blood vessels among the great muscles of the leg corresponds in most cases to the clinical signs of arterial deficiency, that is, to the severity of an intermittent limp. By contrast with the abundant net

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or female, perhaps more often a male, but thrombo anguitis obliterans in a female is almost unknown. An arteriosclerotic deficiency is either so well balanced by a collateral circulation as to cause only minor disorders to which the individual gives little attention or it develops rather rapidly into a serious local or extensive gangrene. Perhaps only one individual among ten who show minor signs of arteriosclerotic deficiency ever comes to ulceration or gangrene, but that one may suffer from an extensive necrosis of the toes or a foot after a very short period of premortory cyanosis and pain. By contrast, the pre-gangrenous stage of Buerger's disease may be prolonged for years, and its actual gangrene need never be very extensive, which is another way of saying that symptoms of circulatory deficiency threatening ulceration or gangrene are seldom noticed in arteriosclerotic disease for more than a few months, but in thrombo anguitis obliterans may be present for years. Again, though arteriosclerotic deficiency, as between the legs and arms, shows itself almost exclusively in the legs, there are often telltale signs of arteriosclerosis elsewhere, notably dizziness, transient aphasia, lack of tolerance for cold, and the arteriosclerotic vessels appear calcified to the X ray. By contrast, the signs of thrombo anguitis obliterans are noticeable chiefly in the legs or only very late, in the arms, and calcification of the arteries is rare and much delayed. In earlier times, perhaps before cigarette smoking became general, it seemed that thrombo anguitis was confined to Polish or Russian Jews. And though such is no longer true, the disease, in the United States at least, is seen more often in Hebrews than in any other race. Finally, arteriosclerotics never suffer from superficial "wandering" phlebitis, whereas some observers have maintained that phlebitis migrans attacks those suffering from Buerger's disease in as much as thirty per cent of all cases.

**The Presenting Symptoms**—Intermittent lump, if the story could be dragged out of every patient, would probably be found to be the most common initial symptom of arteriosclerotic deficiency. But among those who present themselves for

work of very fine arteries and arterioles so often seen in thrombo-angutis obliterans, the individual arterial branches are fewer and more irregular in caliber. In both diseases, the total arterial circulation may be equally lacking and the functional difficulty much the same, but in arteriosclerosis the way is laid for ultimate gangrene on a larger scale. The clinical application of these characteristic pathologic changes will again be discussed when the course of the two diseases is compared.

There is a pathological variation upon arteriosclerotic deficiency known as *Monckeberg's arteriosclerosis*. This is characterized by sclerosis of the media rather than the intima, is thought to occur a little earlier in life than the common form, and is not so apt to exhibit calcification to the X ray. However, it may not be an independent process but rather a stage of the usual disease, and that it can be distinguished as a clinical entity is exceedingly doubtful. Its supposed peculiarities will be considered with the clinical manifestations of arteriosclerotic deficiency and in particular with the differential diagnosis between this state and thrombo-angutis obliterans.

Arteriosclerosis and thrombo-angutis obliterans include ninety-five per cent of all arterial deficiencies. Of this percentage arteriosclerosis has much the larger share. Both diseases produce their effects by arterial narrowing or obstruction, so that they necessarily have a family resemblance. It is in their background and in the progression of their signs and symptoms that the two diseases mainly differ. And though it may happen that at any one moment in the course of each, and in the unusual individual whose age, sex, and race are consistent with either disease, a differential diagnosis is difficult, the etiological factors contrasted in the following paragraph will greatly aid in making a distinction between them.

The victim of arteriosclerotic disease is rarely under fifty years of age, the average of those who first complain of symptoms being perhaps sixty, in contrast with thrombo-angutis obliterans which usually shows itself between the ages of twenty and forty. The arteriosclerotic may be either a male

haps the forefoot become red or cyanotic in the dependent position and that some particular toe is discolored before the others. He may notice also swelling, which is the next step in the self revelation of the deficient circulation, but these are rather objective signs to be noted by the observer.

The Clinical Signs of arteriosclerotic vascular deficiency, in the absence of acute arterial thrombosis or actual gangrene, again are suggestive rather than striking. Especially in the very elderly, there is almost always some atrophy of the legs, most obvious in, but not confined to, the calf. The surface of the leg, foot and toes is dry and cool. The skin is often rather thin and tending to be transparent. Such a state is, of course, consistent with a deficiency of long duration and is seldom a cause of complaint. But when gangrene is imminent or is actually present, the natural color of the skin will usually be changed. Then some one or several of the toes may be red or even bluish red. The discoloration may run up upon the foot itself. By contrast with thrombo angitis obliterans, such redness or cyanosis is less common and when present, is less well marked. Edema of the toes and forefoot is likewise less important in arteriosclerotic deficiency than in Buerger's disease—a difference of degree only and not a reliable distinction. Both cyanosis and edema are likely to come out best when the foot is left dependent for considerable periods. Should spontaneous pain be present it will likewise be increased by letting the leg hang.

*Minor Gangrene*, in the absence of diabetes, which will be discussed in a separate section, is usually well localized. In the type which follows a slow atrophying sort of arterial occlusion and in which a gross occluding thrombosis is not a factor, it will often have begun beside the great toe nail, as already described, involving part of the outer phalanx, but will not reach the base of the toe. Or if one of the middle toes is particularly deformed or calloused, a local ulcer associated with very little gangrene may have formed. A callus over the prominent metatarsal head at the base of the great toe is a common site of gangrene and a corn upon the little toe is



the treatment of serious pain, ulceration, or actual gangrene, it is not the usual presenting symptom, which actually is numbness and coldness of one or both feet. Very likely, the state of the arteries has long prevented the individual from walking any considerable distance or at anything more than the slowest pace. Yet having been broken slowly to this situation, he, or she, seldom thinks to complain of the cramp like numbness, excited by locomotion, which disappears so quickly on rest. See many an elderly woman on a street crossing. It is not necessarily stiff joints which makes her move so slowly. She just can't walk faster and knows it. Only if she hurried would she exhibit the limp of arterial deficiency. Intermittent limp, already touched upon, will be more fully described in the chapter devoted to thrombo angustis obliterans.

Beside the characteristic feeling of coldness, of tingling, and of a cottony sort of numbness, there is sometimes a localized pain in the foot principally affected, a pain which comes and goes, at first, without much reason but which tends to become constant. It may be felt in the sole, the instep, the heel, or in the toes. Such a pain or a feeling of coldness of the feet may prevent sleep at night, but even if such troubles are absent, night cramps frequently occur. These are apt to be confined to the foot or to some particular group of muscles of the leg, often causing violent dorsiflexion of the great toe or all the toes.

Trophic changes are common, cracks, especially upon the heel, and thickening and deformity of the nails. This last may result in unskillful cutting of the nails or of attempts to treat what seems to be, but probably is not, an ingrowing toenail upon the great toe. An individual often complains of soreness from the irritation of a deformed nail and ends by developing gangrene of the great toe as a result of the trauma and infection brought on by an ill advised attempt at a surgical cure. Indeed, treatment of the corn, callus, or nail with the knife is very commonly a beginning of the familiar series of events, infection, ulceration, and gangrene.

The patient is occasionally aware that the toes and per-

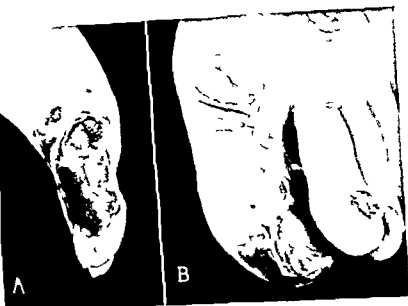


ARTERIO-LEPOTIC CANCER (1 R K) a mild diabetic sixty-eight years of age symptoms of only a few weeks duration local gangrene absent peripheral pulse calcified vessel The limb was finally cut off with healin B J B sixty year old age symptoms of few weeks duration local gangrene healed formed nail absent peripheral pulse calcified vessel Recovered under local treatment of nail and wet dressing C Most Type line to thrombosis I N symptoms for two years cyanosis and numbness for three weeks leucocytosis beginning

especially likely to touch off an ulcerative, gangrenous process. With all such small lesions there will sometimes be found necrosis of a phalanx, a process slowly suppurative and detectable by the X ray but hardly worthy of the name of osteomyelitis. A sinus may lead down to such a spot or to an infected joint, but as compared with similar states in diabetes, there is little redness and tenderness, that is, external evidence of infection. Tendons and tendon sheaths are relatively seldom involved.

Any area of gangrene, if seen from the beginning, will first appear a deep purple, turning slowly to black. Not for a week or more will the border of such an area become clearly defined, but as demarcation proceeds, the gangrenous part will shrink and dry. Meanwhile, the adjacent skin is becoming a little red and swollen. The point is often made that when the outer half or all of a toe is gangrenous, the process cuts just as definitely through the deeper part as through the skin and should be allowed to take its own pace, so that final separation, which only takes place after several months, is given the opportunity to leave a granulating surface capable of healing. The proximal zone of reactive hyperemia and induration is in the meantime a protection against infection. To amputate the gangrenous toe of arteriosclerosis is very hazardous and only those thoroughly familiar with the disease should ever attempt it. Nature's method is slow, to be sure, but safe.

There is one rather notable distinction between the gangrene of arteriosclerosis and of thromboangitis obliterans. Pain in established arteriosclerotic gangrene can seldom compare in severity with that of Buerger's disease. There is a burning, agonizing quality, sometimes a feeling as if the toes were being crushed, and an associated hypersensitivity in the gangrene of Buerger's disease which is very rare in the purely arteriosclerotic states. Often the gangrene of arteriosclerosis will be sore rather than painful, though elevation and occasionally hanging the foot down may bring on considerable pain. Absence of pain is even more marked in diabetic gangrene as will presently appear. In such, the gan



**ARTERIO-CLLOTIC GANGRENE - I I K C** a mild diabetic sixty-eight year of age symptoms of only a few weeks duration local gangrene absent peripheral pulse calcified vessel. The limb was finally cut off with healing. **B I I B** sixty year of age symptoms of one weeks duration local gangrene little deformity nail absent peripheral pulse calcified vessel. Recovery under local treatment of nail and wet dressings. **Mort** Type due to thrombotic. **I N** symptoms for two years cyanosis and numbness for three weeks demarcation beginning.



grenous, perhaps infected, part is often actually anesthetic. An interphalangeal joint, for instance, so far destroyed as to grate on motion, will often cause no pain whatever

One form of senile arterial disease, undoubtedly arteriosclerotic, behaves so much like thrombo angustis obliterans as to be easily confused with it. The patient is in the forties or fifties rather than the sixties, that is, prematurely aged. The threatening prodromal symptoms develop more slowly than is usual in arteriosclerosis though rather too rapidly for thrombo angustis obliterans. The cyanosis of the toes and forefoot or it may be the actual gangrene, is particularly painful and very resistant to treatment. Apparently a collateral circulation is not readily developed. In such cases, the arteries of amputated limbs have so often shown the medial type of sclerosis characteristic of Monckeberg's arteriosclerosis as to suggest the existence of a clinical pathological entity. However, the matter is hardly as definite as that. It is safer to say that here an arteriosclerotic deficiency behaves like thrombo angustis obliterans but the patient is rather too old for that disease. After all, arterial narrowing is arterial narrowing, and Buerger's disease is chiefly distinguished by its inflammatory background, the involvement of veins, and especially the youth of the subject, which permits collaterals to be opened while the arterial circulation is still elastic. There is no reason why there should not be borderline cases.

*Major gangrene*, often of great extent, occurs particularly when the patient is driven to seek help by thrombosis which suddenly closes a good sized artery, previously patent. Here the duration of symptoms preceding the actual onset of gangrene is especially short. The patient is usually struck down by severe pain, the pain of sudden ischemia, much like that of an arterial embolism. That this varies in severity from a feeling of numb coldness to real agony is well known. In many such cases the whole forefoot, or the whole foot, or the foot and part of the leg will turn pallid, bluish, and finally purple, the color being rather splotchy and with no clear cut limit above. The skin over such an area will be cold, the tis-

sues swollen and perhaps blistered. These signs often, though by no means necessarily, lead to a moist gangrene. In any case, there is a distinct difference between this picture and that of the slower mummifying process. Given infection and edema, a moist type of gangrene occurs. Yet there are many intermediate stages, making a distinction between "dry" and "moist" gangrene impracticable.

**Diagnosis**—The identification of an arteriosclerotic deficiency or actual gangrene rests upon the age of the individual, an appearance of atrophy, the state of the skin, including its coolness, and the evidence of restricted circulation revealed by the simple tests outlined in the preceding chapter. That is to say, it can be shown that the leg and foot are not only cool to the touch but are very slow to become warm when exposed to a heated environment and cannot be made to exhibit anything more than the slightest reactive hyperemia by the various methods described. These, of course, are qualities consistent with an arterial deficiency of any sort, but in addition, the patient, who will seldom be under sixty years of age and usually a good deal older, will rarely complain of having suffered from coldness, numbness, or an intermittent limp for more than six months, and if actual gangrene is present, for a far shorter time, whereas the much younger individual suffering from *thrombo angitis obliterans* will almost necessarily have noticed an intermittent limp for years. The physical signs are confirmatory. The toes and foot seldom become as cadaveric on elevation or as cyanotic on depression as in the correspondingly serious case of Buerger's disease. Moreover, the larger gangrenes involving the foot and part of the leg and due to a rapidly developing thrombosis are far more extensive than are ever occasioned by the latter condition.

Pulsation in the larger arteries often reveals a difference between the two legs but such pulsations are not to be counted on to distinguish arteriosclerosis from other deficient states. A pulse in the *dorsalis pedis* or posterior tibial artery is fairly often present in the face of a decided intermittent limp, signs of coldness and atrophy, and even actual gangrene. But all

four arterial pulsations are never palpable Both posterior tibials will be absent and the dorsalis pedis of the leg most seriously affected will perhaps be feeble Or all pulsations will be missing on the worst side and only barely detectable on the other This is in contrast with thrombo angustis obliterans where peripheral pulsations are almost always absent once clinical signs of the disease have appeared Sometimes in the presence of diabetic gangrene, which after all is merely arteriosclerosis with or without infection, the pulsations of several peripheral arteries are remarkably vigorous In other words, arteriosclerotic deficiency is a patchy one, the supply to some parts being good and to others poor

**Methods of Recording and Classification**—By this time, sufficient information has been presented, in this and the preceding chapter, upon which to base a method of study and system of record. And though arterial deficiency has chiefly been discussed, the inevitable comparison with thrombo angustis obliterans will have given at least a partial account of that disease Accordingly, a table of differential diagnosis is presented below and, following this, a scheme for making special notes in vascular disease Both of these are taken in great part from A. W. Allen's publications which in turn are based upon procedures devised in the Vascular Clinic of the Massachusetts General Hospital The writer has taken some liberties with both the table and method of notation Monckeberg's Arteriosclerosis has been omitted, on the ground that it is hardly a clinical entity Vasomotor disorders, as a matter of contrast, are included though, strictly, they are too little of one type to be covered in this way It should be realized that the etiologic, pathologic and clinical signs recorded often represent averages and tendencies rather than fully reliable indications of disease

### Special Notes

*The first seven headings relate to the history Headings 8 to 10 relate to diabetic gangrene The final group of six relate of course to special examinations and are not equally required*



TABLE OF DIAGNOSTIC INDICATIONS

	ARTERIOSCLEROSIS	THROMBO-ANGITIS OBLITERANS	VASOSPASM— RAYNAUD'S AND PERMANENT FUNCTIONAL TYPES
<i>Age</i>	60+	20-40	15-30
<i>Sex</i>	Males and females	Males	Females
<i>Nationality</i>	All	Hebrews 40% All but negroes	All
<i>Duration of Symptoms</i>	Months	Years	Years
<i>Extremity</i>	Lower	Lower (until very late)	Raynaud's upper Permanent type lower
<i>Symmetry</i>	Unilateral or one side at a time	One side at a time	Bilateral
<i>Ulceration and Gangrene</i>	Early after declared symptoms	Late	Late and moderate
<i>Vessels in X ray</i>	Calcified	Not calcified	Not calcified
<i>Peripheral pulses</i>	Poor or none	None	Normal
<i>Procaine Block</i>	No vasodilatation	Slight vasodilatation	Vasodilatation
<i>Collateral Circulation</i>	Present but erratic	Many fine vessels	Not present or required
<i>Pathological State of Vessels</i>	Thick irregular in tima, atheroma and calcification	Inflammation, organized thrombosis—arteries and veins	In Raynaud's disease only, then late sclerosis of digital arteries

for all cases They are fully described in the previous chapter

1 The duration of symptoms intermittent limp, coldness, numbness, trophic changes If ulceration or gangrene is present, its preceding and prodromal symptoms the duration of ulceration or gangrene

2 Date and character of injury or local treatment as an exciting factor in the development of ulceration or gangrene

3 Occupation when disability began, how far the individual depends upon the use of the affected limb or limbs

4 Nature and severity of pain circumstances of its onset, its development Is it spontaneous or dependent on the use of the limb?

5 Use of tobacco reaction to other factors such as cold

6 Description of the lesion the present state of ulceration, gangrene and infection.

7 Nature of the peripheral pulsations their presence or absence in the dorsalis pedis, posterior tibial, popliteal, femoral arteries These to be described as + to + + + +

*For diabetics all held to be emergencies*

8 Blood sugar to be studied

9 Blood cultures to be taken if infection is present or suspected.

10 Cultures taken from all open wounds

*Tests to be recorded*

11 Effect of elevating and depressing the leg time required for blanching on elevation, and flush and cyanosis on depression (routine)

12 Reactive hyperemia in response to vasoconstriction Length of time required and completeness as observed in toes (routine ?)

13 Surface temperatures as noted by contact with back of fingers (routine) as recorded by thermocouple, as a reaction to procaine block, peripheral, spinal (optional)

14 O sphygmometry Oscillations in the calf and their changes in response to a reactive hyperemia by any method (optional)

15 Roentgenological study of blood vessels (routine)

16 Photographs (optional)

It will be observed that except for the X ray and the bacteriological investigation, both of which are available in any well equipped hospital, all the important routine observations require no special apparatus. Such are described in Chapter I, and their relative importance is there discussed.

#### **Treatment in the Absence of Ulceration and Gangrene—**

For the individual who complains of a deficient circulation without gangrene—numbness or coldness, intermittent limp, perhaps discoloration of the toes, nocturnal cramps—there is a useful routine which can be expected to lessen the patient's discomforts, retard his downhill course, and, in the younger and more elastic group, lead to actual improvement. This depends upon the development of collateral vessels to compensate for the gradual closure of one or more of the larger peripheral arteries—the anterior and posterior tibial and the peroneal. It should be comforting to perhaps 90 per cent of such persons to know that by giving up tobacco, curtailing their physical activities, and encouraging their circulation by vascular exercises they will be able to live in comfort and avoid the ulceration or gangrene which may have seemed to threaten them.

*Tobacco*, if the patient is a smoker, must be barred. It has nearly as unfavorable an effect upon arteriosclerotics as upon those suffering from thromboangitis obliterans. It is especially to be avoided in those having pain, whether brought on by exercise or of a spontaneous sort. And since smoking definitely causes vasoconstriction, it may be expected to interfere with the widening of the vascular bed and the development of a collateral circulation, the first objects of treatment.

*Fatigue* should be relieved. This may mean rest in bed for the tired housewife or a vacation for the business man. The laborer may have to devote his days off to reclining or actually going to bed, the feet being elevated to the position of greatest comfort, which may mean the horizontal or a little below it. For the indoors person, the stimulation of outdoor air and

such mild exercise as can be taken without exciting discomfort is a definite advantage. Though many individuals who complain of a deficient circulation have a high blood pressure, some have a low one, in which case the delivery of blood through sclerosed arteries will be aided by any treatment which increases the force of the heart beat. Hence proper rest, gentle exercise, and an outdoor life will be doubly effective.

*The Routine Care of the Feet*—This is of great importance in arteriosclerotic deficiency and becomes even more vital in the presence of diabetes. After a daily bath in warm water and careful drying, olive oil or lanolin should be rubbed into the skin, particularly where cracks are present, as upon the toes or heels. A search should be made for scaling or blistering suggestive of epidermophytosis which may become a point of entry for pyogenic infection, and so lead to ulceration and gangrene. If the fungus is found, its treatment is a problem. Usually an ointment, such as half strength Whitfield's, with thymol 1.5 per cent, is helpful but some patients can be treated successfully only with liquid fungicides, as for instance, 1 to 2000 3000 permanganate solution, five per cent aluminum acetate, or one to two per cent salicylic acid in fifty per cent alcohol.

The toe nails, unless they are grossly thickened and deformed, should be cut squarely and rather long. They should always be soaked before cutting and if curved and thickened may be filed. Corns, which are so often a starting point for gangrene, should be pared down by someone expert in that line. Like the nails, they must first be softened by soap and water. Calluses should be reduced with sandpaper.

The matter of a covering for the legs is most important. Wool for socks or stockings is best. If the surroundings can not be made sufficiently warm, lamb's wool casings for the legs at night preserve the natural heat as well as anything. Bed socks at least should be worn on cold nights. Never letting the feet be exposed to cold or actually feel cold is a constructive step in developing a collateral circulation.

Postural exercises, which will be more fully described in

the treatment of thrombo angutis obliterans, are very well worth carrying out in arteriosclerotic states. They are especially useful when discoloration of the toes and signs of edema indicate that the compensatory circulation is insufficient. The physician must work out with the patient the proper routine. It will be found perhaps that the legs blanch in a minute and a half when elevated to an angle of  $30^{\circ}$  ( $45^{\circ}$  might be too high) and that a flush is slow to appear when the legs are allowed to hang, but that perhaps two minutes of depression will secure the maximum pink flush without cyanosis. During the period of depression the feet should be exercised as directed by Allen (page 95). Finally, the legs should be wrapped in warmed blankets and kept horizontal for about five minutes. The physician must devise an inclined plane for elevation, must discover how many times the cycle shall be repeated without too much fatigue and how many times a day a set of individual cycles shall be carried out.

A hot sitz bath, taken once or twice a day at a temperature which feels comfortably hot to the patient, about  $100^{\circ}$ – $110^{\circ}$  F., will perhaps cause satisfactory hyperemia. Such a bath should not be taken for more than ten minutes and the individual should see that the body and legs are not afterwards cooled. It will be found perhaps that the sitz bath works best preceding a period of exercise or that it should follow one before the patient goes to bed.

Diathermy will be used by those familiar with it, but since it calls for a special apparatus and involves an expense over and above that of the simple and useful measures already described, it must be regarded as a part of hospital (or office) rather than ambulatory, or home, treatment.

Drugs, except so far as they may strengthen the action of the heart, improve appetite, assist sleep or relieve pain, are of no real advantage in the treatment of arteriosclerotic arterial deficiencies.

*Treatment of Pain*—If the patient suffers from spontaneous pain or if necessary exercise causes a serious intermittent limp, special treatment over and above the routine out

lined will be required. But it will first be necessary to discover (1) what actually will relieve pain and (2) whether an effective hyperemia in the limb can be induced. These two considerations can usually be counted as one.

The relief of pain by drugs can be dismissed as something not going to the heart of the matter. Morphine especially is very dangerous (and useless).

A case in point is the following. M G L, a man of sixty, had been unable, for many years, to walk at a normal pace without bringing on a cramp like pain in the front and outer side of the right leg. For some two to three years he had noticed a very similar crampy pain at night. The nocturnal pain had recently increased in frequency and severity. For the last year he had suffered an oppressive (anginal) pain in the mid-thorax on exercise. This had been benefited by vasodilating drugs.

The patient looked more than his age. He was reasonably well nourished. His legs were only very slightly atrophied. No arterial pulsations could be made out below the knees. There was a good pulsation in the left popliteal artery, none in the right. The right femoral pulse was less strong than the left. Both feet were cool.

Upon elevating both legs for two minutes, the characteristic discomfort in the right calf set in. Both feet also became very pale and on subsequently letting them hang down, the color came back more slowly in the right than the left, not reaching the toes for perhaps half a minute. The X ray revealed faint arteriosclerotic changes in both femorals and definite calcification not only in the dorsalis pedis arteries, but also in the small vessels running to the great toes. While under observation, the patient was given two ounces of whiskey for his pain one night, apparently without relief, yet he did not walk the floor as usual and soon went to sleep.

In a rather warm room,\* that is, at a temperature of 78° F (25° C) the right great toe was found to have a temperature

\* Naturally the test should have been made in a cool room. Unfortunately none was available. Yet the test was reasonably informative.

of 87° F, the left 88° F and in response to a right lumbar block by the injection of procaine, the temperature of the right great toe rose only 2° F, never catching up with the left, which also rose a trifle. However, the right foot acquired a sensation of warmth, as compared with the left, and elevation for five minutes, while the block was effective, failed to bring on the characteristic pain. Thus the pain was for the moment benefited, yet with only negligible vasodilatation, a state of things which might have been surmised from the simpler observations already made.

In such a case as this, over and above the usual Buerger Allen exercises and the routine protection from cold and trauma, a prolonged trial of suction and pressure or, perhaps better, intermittent venous compression should be made. So little is to be expected from a lumbar sympathetic resection as to make this procedure inadvisable, particularly in view of the cardiac symptoms. There is little danger of gangrene, the problem being to control pain. Drugs will be ineffectual.

In the above account it will have been noticed that a right lumbar sympathetic block gave comfort, bringing up the question whether, if routine measures had failed, injection of alcohol into the region of the right upper lumbar ganglia might not properly have been tried. Both Flothow and Reichert recommend such a course and it may therefore be proper at this point to describe the procedure.

The patient being placed horizontal, lying on the side opposite to the one into which the injection is to be given, a wheal is made six to seven cm lateral to the second\* lumbar interspace, as in the diagnostic block by procaine (page 27) and after the usual infiltration with procaine of the intermediate sensitive structures, the eleven cm needle is directed, at an angle of about 45°, toward the body of the second lumbar vertebra. A second needle is passed toward the third lumbar

\* The usual directions are to inject alcohol beside the first second and third lumbar vertebrae but injection of only L 2 and 3 should secure the desired result and there is a theoretical objection to destruction of the first lumbar ganglion in the male.

vertebra, the plan being to see that the points of both needles are well anterior, that is, toward the front of the body where the sympathetic ganglionic chain is situated. After the usual suction, to make sure that no blood vessel is entered and that no cerebrospinal fluid flows, a few drops of ninety five per cent alcohol had better be injected into each needle. If the points are well placed, a transient epigastric or abdominal pain will be felt. In the absence of this sensation it is almost certain that the needle points are not near the ganglionic chain and they should accordingly be shifted to a more satisfactory position. Once the pain appears, two to four cm. of ninety five per cent alcohol are slowly injected. In any case, the fluid should be kept well in front of the lumbar spinal nerves, lest an alcoholic neuritis, which may last for a month or two (almost as serious a matter as the original complaint) be set up. Indeed this is the real objection to the procedure which, in many cases, gives a high degree of relief. The first effect of an accurate injection about the ganglia is a severe pain of a few minutes' duration, due to a preliminary irritation of the ganglia and associated with sweating and coldness in the extremity. Following this, a feeling of warmth, both subjective and objective, comes on.

It has been said that smoking in arterial deficiencies should be forbidden. There is perhaps no other routine measure so likely to relieve pain. In the following case, there was only a minor complaint of spontaneous pain, but the intermittent cramp and such spontaneous discomfort as was present were done away with in an almost miraculous way.

S.D.F., a man only fifty years of age, a heavy cigarette smoker and inhaler, had noticed for two years, on walking even for a short distance, a feeling as if a knife had been thrust into the calf of his left leg. This disappeared in the usual way on rest. The trouble had since advanced so rapidly that recently he had been able to play continuously no more than three holes of golf. At the same time he had begun to wake in the morning noticing numbness in the instep and the great toe of his left foot. The latter became cold very easily.



The patient looked rather more than his years. His blood pressure was rather low (118 systolic). There was an obvious atrophy of the calf muscles. No arterial pulsations could be made out below the femorals. Both the feet appeared cadaveric on elevation for two minutes and flushed slowly on depression, the left toes requiring twenty seconds before showing color. On passing one's hand down the leg, a faint but unmistakable change from warmth to coolness could be felt a little above the ankle. The feet, however, were dry and not noticeably cold.

The patient was given postural exercises and directed to stop smoking. In two months a decided change had occurred. He could now walk slowly on soft ground for two miles without having to stop because of cramp. He no longer noticed numbness of his left foot on waking. A faint pulsation could be detected in both the left and right dorsalis pedis arteries, the left the stronger. The flush on hanging down the leg after two minutes' elevation passed out upon the toes of the left foot in seven seconds instead of twenty. In three months more the patient's cramp had so far diminished that he could walk fast for perhaps a quarter of a mile. Then the limp returned. He could play eighteen holes of golf.

Here is an evident arteriosclerosis with a strong suggestion of superimposed vasoconstriction due to the abuse of tobacco. Giving up cigarettes vastly relieved the intermittent limp (postural exercises probably helped) and the peripheral pulses returned, but once this change had occurred, the smaller vessels were found to have little capacity for reactive hyperemia. Spinal anesthesia caused the temperature of the great toes to rise moderately, the left  $9^{\circ}$  F and the right  $4^{\circ}$  F but in neither case was the low normal level of  $90^{\circ}$  F ( $31.5^{\circ}$  C) reached.

**The Treatment of Gangrene**—Since pain in arteriosclerotic states is not usually of great importance, the first consideration is the limitation of gangrene, the second, is the securing of healing once the gangrenous part is cast off or removed, and the last, of course, is the problem of amputation. In most

instances, gangrene is of the dry or mummifying type, and ulceration is limited and uncomplicated save perhaps by a local necrosis of a phalanx or disintegration of a joint. In the absence of diabetes, infection is seldom serious.

Lamitation of gangrene calls for rest in bed. The foot should not be elevated, since arterial blood will then reach the toes with even greater difficulty than is already the case. On the other hand, if the leg is too dependent, edema will occur. It will usually be true that a horizontal position for the legs and a reclining one for the body will give about the right amount of moderate venous congestion and of increased capillary pressure. The foot should be protected from trauma by a large cradle which crosses the entire bed and includes at least the full length of the legs. The cradle should not, however, be heated or if heated the temperature should merely be warm, that is, not over 80°-90°F. For local heat calls for a more active metabolism than the crippled arterial system is able to support and if it does not actually burn the exposed parts, it does them harm (increased anoxemia) rather than good. The leg had better be kept warm in wool or cotton.

The actual dressing of the foot should be made an aseptic ritual. The healthy skin is best gone over with a neutral soap, using sterile cotton pledgets held in a sterile instrument, then wiped off with sterile water, or seventy per cent alcohol. Finally, when the gangrenous area has been treated, the near by skin should be covered with vaseline gauze. For the gangrenous part itself every one is apt to have his favorite remedy. Once securely mummified, its covering, except it be protective and clean, is unimportant. In the earlier stages it may be patted with any mild antiseptic and covered with gauze moist with the same solution. Alcohol, seventy per cent, is useful for its drying quality. The truth is that routine cleanliness is more important than any drug. But if infection makes it seem advisable to keep the dressing moist, one must choose some solution. Samuel warmly recommends soaks of 0.5 per cent watery chloramine solution to aid in the separation of small sloughs and to clean ulcerated areas. A watery

iodine solution (Lugol's solution 1-400) or a coconut oil derivative (alkyl dimethyl benzyl-ammonium chloride) in a strength of 1-1000 are nonirritating and almost as good dilute antiseptics as any "Eusol",\* mixed, equal parts, with mineral oil, is an excellent antiseptic and surprisingly little irritating to the skin

*Local Amputation*—Once the gangrene is localized and a zone of reaction established proximal to the line of demarcation, or once it is evident that a sinus leads down to a disorganized joint or an area of necrotic bone, the problem of getting rid of the necrotic part and securing healing arises. The safest and most time consuming method is to allow a gangrenous toe to be cast off. For the tip of the toe this is all very well, but for half a great toe, for a whole smaller one, or for a necrotic phalanx, local amputation, provided certain rules are observed, is economically desirable and may occasionally be performed. However, it is almost criminal to amputate such a toe in supposedly sound tissues proximal to the area of reaction. Amputation, to be even reasonably safe, must be made very close to the border of the gangrene in the red zone of reaction. If the tissues are cleanly divided and not traumatized and if any exposed phalanx is cut across, not disarticulated, the wound can be left open with good prospects of healing. Nature has already set up a defense against infection. The operation asks little new of the patient.

It is at the stage when gangrene is over and granulation plus epithelization are starting that the details of treatment will decidedly help or hinder. In well equipped institutions, the Carrel Dakin technique, skillfully used, is effective. But treatment of an open wound lined with a thin slough in avascular tissues may include anything from the use of Dakin's fluid to pure urea crystals. When a wound is left boat shaped, as after the necessary removal of a necrotic metatarsal head,

* The formula for Eusol is	
Boracic Acid	12.5 grams
Chlorinated Lime	12.5 grams
Distilled water	1000 cc m

Mix allow to stand over night and filter

the use of Dakin's fluid or dichloramine T (eight per cent in chlorcozane) or Eusol and mineral oil are especially recommended. Self draining wounds are easier to treat.

Once healing is under way, an attempt to secure a reactive hyperemia and a permanent widening of the vascular bed are apt to bear fruit. Rhythmic suction and pressure, or venous compression, will often hasten healing and of course the routine treatment as for the pregangrenous stage of arteriosclerotic deficiency should by all means be resumed. Whether or not any attempt at a permanent block of the sympathetic supply to the limb shall be made is a matter to be decided on the ground of the patient's proven capacity for vasodilatation and the probable estimated future of his circulatory deficiency. On the whole, little is to be expected in arteriosclerotic gangrene from sympathetic resections.

*Amputation of the Limb*—It will be profitable to discuss the criteria for the amputations at any level above the toes in connection with diabetic gangrene at the end of this chapter. Amputation is demanded when so much of the foot is destroyed as to make it useless, when a life is to be saved in the presence of uncontrollable infection and, very rarely, on account of pain or for economic reasons. Amputations below the knee can almost never be expected to offer a healthy stump. Most are performed through the knee joint or through the lower third of the thigh.

### Thrombosis in Arteriosclerotic Deficiency

Doubtless large vessels already considerably narrowed at some one point can finally be closed by thrombosis without bringing on any sudden or noteworthy change in the circulation of a leg. The rapid closure of an artery carrying a good blood supply is a very different affair. Except that no source of embolism is apparent—that is, the heart is not fibrillating and nothing resembling a coronary infarction has occurred—the suddenness of the ischemia much resembles that of embolism. There will have been perhaps no particular warning, no premonitory numbness, coldness, or limp. The pain of a

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sudden thrombosis is usually severe, sometimes agonizing, but as already explained may take the form merely of numbness or coldness. In any case, it is of such a character that the patient is immediately driven to seek help. The following is a case in point of a mild sort.

C. L. C., a woman, seventy seven years of age, having previously considered herself well, was seized rather suddenly, three days before coming under observation, with a severe pain in the left leg, radiating downward into the left calf and foot. Since then, a burning sensation had settled upon the dorsum of the foot. This pain was particularly bad at night, keeping the patient awake as long as the leg was left horizontal in bed but being relieved when the leg was hung out of bed, or when she hobbled about. Twenty four hours before entering the hospital, the burning pain spread to the heel. There had been no cramps.

Examination showed a rather frail old woman, evidently suffering. On exposing the legs in a cool room, the right foot retained its heat fairly well. The left foot cooled rapidly and noticeably, as compared with the right, up to a point some three inches above the ankle joint. When the legs were allowed to hang straight down, the toes of the left foot soon turned reddish blue (the right also to a lesser degree) and this color gradually faded out upward toward the ankles. No pulsations could be felt over either dorsalis pedis artery. There was a questionable pulse in the right posterior tibial—practically speaking no pulsations in either leg below the femorals at the groin. The X ray revealed calcification in both posterior tibial and dorsalis pedis arteries.

The greatest amount of comfort was secured by allowing the old lady to sit in a chair, the legs resting on a stool at a slightly lower level. In this position and with only the aid of mild sedatives, she passed fairly good nights, whereas if she attempted to spend the night in bed, she was made sleepless by pain, which was always of the same burning sort and over which drugs, even opiates, had so little control as to be almost totally useless.

Suction and pressure gave considerable additional relief. It was applied, at first rather tentatively, for half an hour twice a day, and with such improvement that it should undoubtedly have been used more freely, but like most elderly women, this one was utterly intolerant of hospital care. She refused any further treatment which necessitated her stay in the hospital.

Upon going home she deteriorated rapidly. Her pain increased, she lost her appetite, and lived only for about a year. She never developed actual gangrene.

Some thromboses cause such sudden widespread ischemia as to be fatal, the effect of the closure being shocking and attended by other signs of circulatory failure and death within a few days. In such cases the foot and half the lower leg often become gangrenous.

Less serious accidents, yet leading to gangrene, may resemble the following.

P N, a woman, sixty four years of age, had been well up to three weeks before coming under observation. Then, rather suddenly, the right leg began to "feel sore" and she found that she must lie down after walking only a very short distance. The right foot almost at once became blue, the toes black. Coldness and numbness "bothered her greatly." Vesicles soon appeared upon the lower third of the leg and foot, increasing in size as the discoloration spread upward.

Examination showed a well preserved woman. No pulses could be felt in the right leg below the femoral, whereas the pulsations in the left leg, including both posterior tibial and dorsalis pedis, were normal.

The right leg, as can be seen in Plate I C was the seat of what seemed to be a moist gangrene. Yet the patient was treated conservatively, sterile dressings being applied, and on leaving the hospital about a week later, against advice, the lower leg and foot were shrinking and the cyanotic, cold area was receding. Thus the threat of infection, especially with the gas producing bacilli, was being removed. The patient has since been lost sight of, yet it hardly seems possible that she



saved her foot. In such a case as this, careful study of the level at which an efficient circulation ended, might have permitted an amputation below the knee.

The fact that gangrene starts in a moist way does not necessarily call for an immediate amputation. Yet the danger of serious infection is such that the leg must be watched intensively. If infection shows itself and spreads upward, an emergency guillotine amputation will be required. With a neat, clean amputation, at or even below the knee, as the reward if conservatism succeeds, and an emergency guillotine amputation, possibly at a high level, the penalty of failure, the difficulty of treating a case of this sort is apparent.

### DIABETIC GANGRENE

Diabetic gangrene is really arteriosclerotic deficiency plus actual or potential infection in a diabetic. The background of arteriosclerosis is only partly the effect of the diabetes (lipemia, deposit of fat in the media of the arteries) for arteriosclerotic deficiencies in diabetics show themselves only a few years earlier than do similar deficiencies in nondiabetics. It is really the lowered resistance to infection, especially by the common pyogenic bacteria of the skin, which gives diabetic lesions of the toes and feet their especially dangerous character. In 1925, Coller and Marsh called attention to the importance of separating the pure arterial deficiencies from those primarily due to infection. Thus McKittrick has followed up, presenting the clinical picture under the heading of "Conditions due primarily to arterial insufficiency" and "Conditions due primarily to infection." This classification will be used here but it should be kept in mind that any but really young diabetics are likely to be arteriosclerotic, and that even in those whose disorders seem typically arteriosclerotic, the tissue cannot be expected to have even the ordinary arteriosclerotic's resistance to infection.

**Conditions Primarily Arteriosclerotic**—The onset of symptoms is not different from that of a nondiabetic, arteriosclerotic deficiency except that the patient may be five years

younger Coldness, numbness, intermittent limp are common complaints The symptoms calling for treatment will seldom have been present for more than a few months, and usually for weeks rather than months The appearance of the toes and feet is like that of arteriosclerosis There will often be left some portion of the peripheral arterial pulsations There is the same liability to closure of a large artery by thrombosis, and there is even more of a tendency to irritation by a deformed nail, to infection of a badly treated corn or callus Thus the routine treatment, outside of that required by the diabetes itself, is the same—encouragement of the circulation in general, preservation of the warmth of the limb, vascular exercises, and in particular, exquisite care of the nails and skin

When gangrene or ulceration or necrosis of some of the toes occurs, it looks and behaves like any arteriosclerotic lesion All diabetics with a poor peripheral circulation are not sitting on powder barrels! Some have a mild disease controllable by diet, and even a condition bad enough to require insulin does not necessarily rob the individual of all resistance to infection The real trouble is that when infection once starts, fixing itself upon a joint, entering a tendon sheath, and in particular progressing along the lymphatics up the foot and leg, there is set up at once a vicious circle of deficient circulation lowered resistance to infection and aggravation of the diabetic state which will often take life if it is not broken by a radical amputation. Undoubtedly, a fatal infection can, though it seldom will, start from a perfectly dry gangrene of the outer half of the toe However, as an example of what a mild diabetic will put up with, and as a lesson in how things should not be done, the following case is quoted

J Z Q, a Jewish male of sixty seven years, had been well up to twelve years before he came under observation At this time he began to have mild anginal attacks for which he was occasionally obliged to take vasodilating drugs At the same period, a diagnosis of diabetes was made and at one time insulin was required Yet in the succeeding years the disease

made so little progress that he was not even very careful to follow his diet. When he entered the hospital his blood sugar was averaging 0.215 per cent, his urine showed only a trace of sugar, and he soon became sugar-free upon diet alone, the blood sugar remaining at 0.146 per cent.

Ten days before entry, the left great toe was noticed to be dark in color, since when a sore, inflamed area had gradually developed upon the inner edge of the great toe nail. The toe had become increasingly painful and tender.

The patient was a fairly well preserved, cooperative individual whose peripheral arteries were everywhere hard. There was a suspicion of an arteriosclerotic aortitis. All pulsations in the right leg, including both the dorsalis pedis and the posterior tibial, were present, those on the left below the femoral were absent (recent thrombosis?). The left great toe was deep red. An area of excoriation was present along the inner edge of the left great toe nail, from which a little pus oozed. Under alcohol applications and hot saline dressings, the cellulitis promptly cleared up. The nail was trimmed square but not short. A small area of granulation was left, but the whole toe from the metatarsophalangeal joint down remained a deep red without actual gangrene.

At this point a mistake was made. Instead of insinuating a little gutta serena tissue or some other nonirritating substance under the edge of the nail and continuing the warm wet dressings, the nail was avulsed under spinal anesthesia. This left still more exposed raw surface, which, during the next few weeks showed no tendency to heal. Here surgical action was again substituted for conservative treatment.

Instead of continuing antiseptic dressings and instituting attempts at reactive hyperemia, which might have encouraged granulation and epithelization, the toe was amputated, not in the zone of redness, but above it, not by dividing the proximal phalanx, but by disarticulating the toe at its base. Result, a sloughing wound, but fortunately no extension of infection. A month later there was evidence of circulation in the skin about the sloughing hole, and a little new skin had grown about its

edge The battle is therefore a draw Very likely the patient's leg will be saved With a greater display of patience his toe might have been!

The subject of conditions due primarily to arterial insufficiency can be summed up by quoting, with explanations, from McKittrick and Pratt

1 No or feeble pulsations are present in the peripheral blood vessels of the leg

2 The foot is cold, blanches on elevation and becomes dusky or red and shiny when dependent Between the mid foot and the knee there is frequently a level at which the cutaneous temperature can be felt to change

3 When necrosis of some part of the bony structure of a toe occurs, gangrene is usually evident

4 Pain is apt to be more severe than the local lesion appears to warrant

5 Gangrene is common

Local amputations in this group are rarely successful and frequently are dangerous Infection in a pulseless foot is the origin of most cases of septicemia Thus, in the pregangrenous stage, the usual routine treatment, as for any arteriosclerotic deficiency, should be used, special care being given to avoiding minor infections about the nails When gangrene is present, the toe or area should rigorously be protected, and in most cases allowed to separate Such treatment is described in a later section

**Conditions Due Primarily to Infection.**—Whether or not gangrene is present, infection in the form of cellulitis and lymphangitis, that is, a streptococcal form, is obvious A favorite initial lesion is the ingrowing toe nail, runaround or ill treated corn or callus One toe, or a toe and part of the forefoot, is red and swollen One or more red streaks of lymphangitis are apt to extend over the foot, upon the ankle and even up the lower leg Pulsations in the vessels of the foot are often vigorous Pain is not a feature and the foot is warm, even hot, partly because the arterial circulation is sufficient but more because of the inflammation To sum up

made so little progress that he was not even very careful to follow his diet. When he entered the hospital his blood sugar was averaging 0.215 per cent, his urine showed only a trace of sugar, and he soon became sugar free upon diet alone, the blood sugar remaining at 0.146 per cent.

Ten days before entry, the left great toe was noticed to be dark in color, since when a sore, inflamed area had gradually developed upon the inner edge of the great toe nail. The toe had become increasingly painful and tender.

The patient was a fairly well-preserved, cooperative individual whose peripheral arteries were everywhere hard. There was a suspicion of an arteriosclerotic aortitis. All pulsations in the right leg, including both the dorsalis pedis and the posterior tibial, were present, those on the left below the femoral were absent (recent thrombosis?). The left great toe was deep red. An area of excoriation was present along the inner edge of the left great toe nail, from which a little pus oozed. Under alcohol applications and hot saline dressings, the cellulitis promptly cleared up. The nail was trimmed square but not short. A small area of granulation was left, but the whole toe from the metatarso-phalangeal joint down remained a deep red without actual gangrene.

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duration about the base of the toe. The flexor tendon was sloughing, the sheath infected and discharging. Though pulsation was present in the dorsalis pedis artery, the X ray showed the local small vessels to be calcified. Under alcohol applications and hot packs, a line of demarcation became clear and there developed a zone of reactive redness proximal to it. The great toe and the partly necrotic head of the first metacarpal bone were then successfully removed under spinal anesthesia, the amputation being carried out close to the gangrene through the red swollen tissues, that is, in the zone of reaction. Result, good healing. However, within two years gangrene of both feet followed, probably because the diabetes was neglected.

**Treatment of Diabetic Gangrene Arteriosclerotic Type—**It has already been stated that gangrene due to arterial deficiency should be treated conservatively, partly to avoid dangerous infection, and partly because local amputations will seldom result in healing, even if no infection follows. Precautions against infection being taken, the gangrenous part will often be cast off. Then, under antiseptic dressings and vascular exercises, healing is likely to occur. If economic reasons and the likelihood that the foot, even if healed, will be disabled urge an early amputation of the leg, the operation should be performed at the knee or in the lower thigh, depending on whether or not the individual is or is not likely to be able to use an artificial leg.

**Infected Type Emergency Treatment—**Provided the nature of the local infection—cellulitis, osteomyelitis of a phalanx, suppurating joint or tendon sheath—offers a reasonable hope that local treatment in the form of hot wet dressings, local drainage or local amputation will be curative, blood should at once be drawn for a study of the blood sugar and to see if bacteriemia can be demonstrated. Cultures should also be taken from the local lesion. Medical and surgical treatment must from the start go hand in hand. The use of insulin should at once be begun. Conservative treatment should be carried out tentatively for a preliminary twelve hour period. If, after twelve hours, local conditions are no worse and the

1 Pulsation in the dorsalis pedis artery is usually present

2 The foot is warm and of good color Even if the heat of the inflammation is not present, the observer's touch will find no marked change from warmth to coolness in passing the hand downward from knee to foot

3 Necrosis of the bony structure of a toe without gangrene is very common

4 Pain is related only to the infection and is usually less than the local condition would lead one to suspect The part may even be remarkably anesthetic

5 Gangrene is found only in the presence of infection or after trauma

The following are illustrative cases

H S C, a male fifty-nine years of age Story of diabetes for one year, to which little attention had been paid For two weeks, a black slough had been present over the right great toe joint Beneath this was fluctuation and a disorganized metatarso phalangeal joint There was redness and swelling of the surface of the foot back to the instep Good pulsation in the dorsalis pedis artery No pain Insulin, fifteen to twenty units a day, barely controlled the diabetes Incision, under gas oxygen anesthesia, showed infection of the deep fascial spaces of the foot and failed to halt the infection Amputation (closed) through the leg, six inches below the tibial tuberosity, was followed by healing but the stump was never satisfactory A better and safer procedure would have been a guillotine amputation through the mid leg followed by a closed amputation through the knee joint (Gritti Stokes or Callander type) Very likely an initial closed amputation through the knee would have succeeded Such operations give a very good stump and the chances are that this patient should be able to use an artificial limb with success

E C, a woman, forty seven years of age, a diabetic for two years taking fifteen units of insulin a day For seven weeks she had suffered from an infected blister on the outer surface of the right great toe During this time the great toe had gradually turned black and finally mummified The blood sugar on entry was 0.178 per cent There was redness and in-

toes (all of which end about opposite the metatarsal heads) These should be entered from a lateral incision as shown in the accompanying sketch

For cavities having no dependent drainage, the use of Dakin's fluid, with all the Carrel Dakin ritual, is very satisfactory Indeed this elaborate technique finds here its greatest usefulness, and the various allied solutions and oily preparations are only slightly less valuable The skin of the foot,

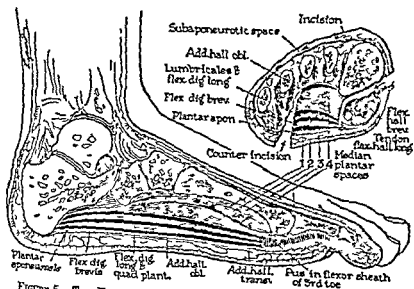


FIGURE 5 THE FASCIAL SPACES OF THE FOOT—(modified from Grodinsky By courtesy of *Surgery Gynecology and Obstetrics*, 49 737, (Dec) 1929 From *Homans's Textbook of Surgery* Courtesy of Charles C Thomas, Springfield Ill., and Baltimore, Md.)

which is so easily injured, must be carefully protected with vaseline gauze No dressings can be allowed to adhere to raw surfaces If no antiseptics are used on self draining wounds, the edges of the skin should be protected with a smooth substance such as gutta percha tissue or vaseline gauze

#### AMPUTATION

Since no artificial knee joint can compare with one's own, the ideal amputation is one which leaves a four to six inch stump below the knee Such a stump, from the very nature of



patient's diabetes seems controllable, a further twelve hour period of observation is permissible. At this time the evidence of the blood culture and the nature of the local infection will be available. The use of sulfanilamide (or an equivalent drug) is very likely to be indicated by the bacteria present. In that case, administration of a full dose of the drug, to secure the optimum percentage in the blood, is required.

If the blood culture is positive, the local lesion is no better or actually worse and the blood sugar difficult to control, a major amputation should be performed.

If, on the other hand, all conditions seem favorable, the local treatment is continued with the idea of performing a minor amputation or operation for drainage at the appropriate moment.

Under such a system, and provided it is thoughtfully planned, local treatment by amputation or drainage will usually succeed. That is, if one third, for instance, of all infected diabetic gangrenes are treated conservatively, only a small proportion of these should require a major amputation to save life or because of failure to secure healing. The other two thirds—the figures are intentionally vague since conditions and skill are variable—will require a major amputation on sight, or at the end of twelve to forty eight hours of expectant treatment, or following failure of a local amputation to halt infection.

Local operations to control sepsis, whether or not they include the amputation of toes, should not be performed in the presence of an ascending lymphangitis, but should await its subsidence.

They should take advantage of every bit of local defensive reaction which warm moist antiseptic dressings, immobilization, and slight elevation are able to produce. If a toe is to be amputated, incisions should be kept away from the sole of the foot. If a metatarso phalangeal joint is infected, the metatarsal head will usually be disorganized and its removal will make drainage more effective. The fascial spaces of the sole which Grodinsky has described are very often invaded by progress of infection from the flexor tendon sheaths of the

*The Temperature of the Skin*—The methods of determining the skin temperature and the inferences to be drawn from such observations are described in Chapter I. In applying them to the problem of selecting a level of amputation, gross evidence is secured first by exposing the previously warmed leg to a cool atmosphere. If one leg from the knee down is cool to the touch, as compared with the thigh and the other leg, any amputation below the knee will be out of the question and even one through the knee joint will be dangerous. Such an observation can of course be confirmed by the thermocouple and by oscillography. A very satisfactory observation is the McClure Aldrich test, which consists in making cutaneous wheals with 0.2 cc. of physiologic saline solution. Normally these wheals should remain visible for the better part of an hour. They are made in series downward from the upper thigh, at four inch intervals, as close to the foot as seems worth while. In the parts poorly supplied with blood the wheal may last only a few minutes. Thus the lowest wheal which lasts more than the half hour marks fairly well the low limit of any proposed skin flap and the bone will of course be divided at a considerably higher level. The wheal can also be made with 0.1 cc. of a 1-1000 solution of histamine (in one per cent novocaine), the normal reaction being a hyperemic flare and a wheal, but this test probably possesses no particular advantage.

*Palpation of the Arteries*—A dorsalis pedis or posterior tibial pulsation suggests that an amputation below the knee will probably be successful. However, it does not guarantee the result. Sepsis, for instance, may forbid the operation, or the patient's history of intermittent limp and numbness of the foot may prove that it is the finer circulation, on which the nutrition of the flaps depends, which is lacking. In other words, arterial pulsation alone is not enough. Other tests must be in harmony.

A popliteal pulsation guarantees nothing for the foot. The main vessels below this point may be defective. Again, not only is the normal pulsation difficult to feel in many persons,

the disease—arteriosclerotic deficiency—for which most of the amputations under discussion here are performed, is almost impossible to secure. Only upon the evidence presently to be described should the mid leg amputation be considered.

The next available level is that of the knee joint. Here amputation can be performed in a field little susceptible to infection, whose skin flaps are rather well supplied with blood from the anastomotic vessels about the joint. The femur is usually divided through the upper part of its flaring condyles and the prepatellar tendon (the patella being excised) or the refreshed posterior surface of the patella itself is used to cover the cut end of the bone, giving what often proves to be an end bearing stump. Amputations of this sort are invariably closed and are intended to be used for an artificial leg having an artificial knee joint. This joint, and indeed the use of the artificial limb in general, requires, on the patient's part, normal sight, normal balance, and normal strength, in fact a moderate athletic ability. And two such legs call for a very able bodied, courageous, youthful person.

The amputation through the lower third of the thigh is a procedure primarily safe. The stump which it leaves will support an artificial limb but very often it is performed with little idea that a limb can be worn.

The guillotine amputation—an emergency procedure to save life threatened by infection—is almost always made in the leg, through its mid-portion, a circular division intended to be left wide open, its surface flat. Such an amputation can rarely be trimmed by a plastic procedure to leave a permanent stump below the knee. Almost invariably it is followed by an amputation through the knee or lower thigh. Rarely a guillotine amputation is made in the lower thigh, with the object, first, of saving life, and second, of leaving a stump which will be closed by a plastic but will probably not be required to support an artificial leg.

**The Selection of a Level for Amputation** is made with the above considerations in mind. The following tests should be used

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A popliteal pulsation guarantees nothing for the foot. The main vessels below this point may be defective. Again, not only is the normal pulsation difficult to feel in many persons,

but the arterial circulation below a slowly closed femoral artery is sometimes remarkably efficient. A more significant finding would be the sudden disappearance of the popliteal pulse, in which case the femoral artery must rapidly have become plugged. With such a change, the clinical signs would undoubtedly agree, as in a case quoted earlier in the chapter, and any other than a guillotine amputation could never be performed below the knee. A feeble femoral pulsation at the groin forbids amputation below the lower third of the thigh.

*Arteriography* has been used in some clinics to indicate the level at which an amputation may be expected not only to heal cleanly but to leave a well nourished stump. Until the skill required for its authoritative interpretation is more generally distributed, simpler methods should be trusted.

**Amputation**—The various operations cannot be described here. It is enough to discuss the present day tendencies. Most amputations below the knee are emergency guillotine procedures, that is, circular divisions performed below an Esmarch bandage. The tibia should be divided four to six inches (ten to fifteen cm.) below its tubercle, flush with the retracted muscle. In the ordinary closed amputation, the fibula would be divided one to two cm. higher but for the guillotine the matter is not important.

For the finished procedure it is a matter of indifference in these days whether the scar is at the tip of the stump or the side. The main thing is to draw some muscular or tendinous structure over the end of the tibia, and the skin flap may well come mainly from the same direction. Thus, the musculocutaneous flap comes best from the lateral and posterior faces of the calf. Bulky muscle should not be used, but its aponeurosis, and perhaps some muscular backing, makes a useful covering for the divided end of the bone.

The amputation at the knee joint, according to the tendencies today, is made at the point where the condyles begin to flare. The skin flaps are rather long. For the Gritti Stokes, the anterior flap includes the skin in front of the tibial tubercle, the posterior flap being shorter. The patella and its tendon are

saved, the posterior face of the former is sawed off, and it is fastened to the open end of the femur. Here it is expected to heal, but does not always do so. If it heals, an excellent end bearing stump results. A rather simpler and more generally acceptable procedure is an amputation at the same level, by which the patella is removed and its tendon made fast over the end of the femur. This gives about as good a stump. The lining of the knee joint is not removed. Callander's operation is of this sort, but it is peculiar in several respects. That is, the prepatellar tendon is merely laid over the end of the bone, the posterior flap is left very long, and nothing is sewed together, the skin flaps being kept from separating only by a few clips. Limbs having a very unpromising circulation can be amputated in this way. The posterior flap draws back, the tendon heals over the bone, and a good stump results.

The thigh amputation exposes large muscular surfaces, which in themselves have little power to heal, but to balance this the skin is now cut in a nearly circular way, so that the operation is not only well away from the infected or potentially infected field, but the superficial tissues are given the ideal opportunity to heal. For no flap can be as well nourished as the skin adjacent to a circular cut.

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## CHAPTER III

### THROMBO ANGITIS OBLITERANS

THIS disease, since its first recognition, has been noteworthy as having a peculiar etiology. It is decidedly more common among Hebrews, especially of Polish or Russian origin, than in any other race. It is nearly confined to males. And it is so greatly aggravated by the smoking of tobacco that really serious gangrene is hardly seen in others than heavy smokers. It is not purely a disease of arteries. This von Winwater recognized in his excellent description of a leg amputated by Billroth for gangrene in 1877. He described a "Peculiar form of Endarteritis and Endovenitis with Gangrene of the Foot", thereby calling attention to the involvement of the veins as well as the arteries and separating the disorder from arteriosclerosis. Since his day, superficial migrating phlebitis, which comes and goes so often with the fluctuations of the disease, has been added to the picture, and finally, in 1903, Buerger, identifying the characteristic pathology with the symptom complex of spontaneous presenile gangrene, gave the name "Thrombo angitis Obliterans".

Buerger holds, "that the disease begins with an inflammatory lesion attended with occlusive thrombosis, and that it affects the arteries and veins in a sort of relapsing fashion, very much in the same manner as in the veins in migrating phlebitis". In other words, it takes the form of repeated attacks, during which new groups of vessels are affected, alternating with remissions, during which organization occurs and a collateral circulation is established. With others, he believes that the acute reaction which the superficial veins exhibit in a minor percentage of all cases, offers the only practical means of studying the earliest stage of the disease, for by the time



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thrombosis were primarily peripheral and tended to extend centrally, a very interesting point in view of present day experience. For many observers feel that the most malignant form of the disease is one which shows itself primarily in the peripheral, or smaller, vessels, as contrasted with a more tractable form which tends to an early occlusion of the femoral itself. It seems to be the peripheral form which causes most intractable pain, is less easily circumvented by a collateral circulation, and is most apt to lead to amputation. Whereas the more central occlusion is not only less painful but is rather rapidly relieved, for the time being at least, by a collateral circulation. Now Buerger's studies were made on the amputated legs of the most malignant type of disease, so that he may not have been describing the commonest form. However this may be, Buerger established the tendency of the disease to attack and gradually close the anterior and posterior tibial arteries and veins, causing them to become adherent to each other and in many cases to engulf in the inflammatory process the associated nerves. And since not only peripheral nerves but sympathetic fibers as well must often be affected, it is easy to understand how the pain of gangrene in thrombo-angitis obliterans is so often far more severe than that of the arteriosclerotic type and why some degree of vasomotor spasm should so frequently be present.

The thrombosis of the early stage of the disease probably results in a greatly diminished caliber rather than permanent obstruction of the arteries and veins. Organization is followed by canalization and the circulation is resumed through narrowed vessels. From a clinical point of view, it is only the arterial disease which is of consequence. Doubtless the extent of the inflammation and thrombosis varies widely according to the severity of each attack in different individuals. But as the years go on, the arterial tree, in the whole limb, is gradually narrowed, the place of its larger vessels being taken by countless fine collateral twigs. Thus it is rare that the peripheral pulses should survive for many years, the upper femoral alone being detectable. This state of things was long ago rec-

the deep vessels can be examined (after amputation) only the late changes can be seen. However, no examination has ever revealed the nature of the etiological factor, though typhus fever, ergot poisoning (from rye bread), and a hypothetical recurring vascular spasm have from time to time been looked to hopefully as causes.

Buerger describes an acute inflammatory process involving all coats of the vessels, and pictures foci containing "giant cells, endotheloid cells or angioblasts and numerous broken down leucocytes", showing actually a purulent area in a thrombosed internal saphenous vein. Yet though various parts of the walls of both veins and arteries have been found in some degree infiltrated with polynuclear leucocytes, most pathologists have been unable to state positively whether thrombosis precedes inflammation or inflammation, thrombosis.

The gross lesion is decidedly thrombosis. To quote Buerger again: "Usually the vessel is seen to be filled with a grayish or yellowish mass that can be distinctly differentiated from the annular wall of the vessel, and that appears to be pierced at one point (more rarely at a number of points) by an extremely fine opening through which a minute drop of blood can be squeezed. Such an obturating lesion is firmer in consistency and does not at all resemble the crescentic or semilunar occluding masses typical of arteriosclerosis. The vessel is usually contracted so that its wall appears somewhat thickened." Apparently the length of vessel involved varies greatly. In the case of an artery, for instance, the process, at its proximal limit, may cease suddenly, the vessel above that point being entirely normal. Below, the red clot may show a long conical end. So far as the veins are concerned, thrombosis is coextensive with that in the arteries. Actually, venous thrombosis is entirely overshadowed by the arterial disease, and so solidly fixed that embolism never occurs.

In some of Buerger's first (eleven amputated limbs) specimens the process extends "from the dorsalis hallucis almost into the popliteal." Indeed he speaks as if inflammation and

*Migrating Phlebitis* is seen in the arms quite as often as in the legs. Indeed it can be studied most easily here. There is an obvious but very mild inflammatory element in every attack. Some vein upon the back of the hand, for instance, becomes slightly painful and sore to the touch. The overlying skin is sometimes reddened. The vein itself, for perhaps two to five cm. (an inch or two) of its course, is noticeably thickened, partly because of hardening of its walls and partly because of adherence of the surrounding soft parts. Yet the skin over the vein is hardly warm. After a few days, such soreness as has been present disappears, and in the course of a week or two the thickened vein itself becomes difficult to feel. At about this time, a new stretch of vein, central to the first but not continuous with it, often becomes sore, or the opposite limb is attacked. Rarely are more than two or three short stretches upon the arm affected in any one period, but it is not so very unusual, in an individual who has become wonted to a migrating phlebitis, to notice the involvement of a vein upon the temple or scalp.

Excision of a vein in the course of a wandering phlebitis reveals the typical inflammation of all the vessel's coats. It is the writer's impression, from a rather limited experience, that the inflammation runs ahead of the thrombosis, which indeed may be insignificant. However this may be, the process finally subsides, leaving behind a vein no longer noticeable and seemingly functioning.

**Visceral Manifestations**—It has been asserted and indeed it appears to be true that rarely and only in the most chronic and persistently vicious sort of thrombo angitis obliterans, thrombosis in some form may attack the mesenteric vessels, giving rise to peculiar and annoying attacks of colic. Apparently the involvement of vessels is not extensive enough to cause gangrene, nor is any particular site for the process recognized. Attention may be called to either the large or the small bowel. There is neither fever nor distension. The colic comes and goes, being excited, perhaps, at one time by eating and at another by defecation!

ognized from the pathological side, but the clinical picture has always been consistent with it and it can now readily be demonstrated by arteriography

Thrombo anguitis obliterans is rarely acute enough to cause early or extensive gangrene. The arterial supply, delivered by countless fine vessels, may be inadequate for an active life, but is not often so deficient as to cause a necrosis of more than one or several toes. For the same reason, the peripheral parts in Buerger's disease have, as compared with arteriosclerotic toes, a considerable resistance to bacteria. They can often be lopped off one by one, so to speak, at a very moderate risk of infection. Long before a spot of gangrene appears under the edge of or beside the great toe nail, that toe and perhaps others will usually have shown the cyanosis, swelling, and shiny atrophic skin of a slow and much restricted circulation. Gangrene of one or more toes and even part of a foot does occur with a fair degree of frequency as the disease advances, but it has been said with a good deal of truth that it is not gangrene which calls for major amputations but uncontrollable pain.

**Thrombo anguitis of the Upper Extremity**—The disease only attacks the arms in the severer cases and after showing itself for many years in the legs. Less is known of its pathology in the upper extremities than in the lower, since outspoken gangrene and amputation of more than fingers or finger-tips are exceedingly rare. However, obliteration of the larger arteries, particularly the radial, does occur, and apparently the small vessels supplying the fingers are rather irregularly involved. The tip of one finger or more will occasionally turn white or cyanotic, showing thickened skin under the nail. Sometimes a whole hand will be affected, becoming bluish and cool. But there is a decided tendency toward the opening up of new pathways and rather more evidence of vasomotor spasm than is likely to be noticed in the toes and feet. Even in the severer forms of the disease, amputation, successfully, of several fingers or finger-tips, without loss of the hand is the worst which the patient is likely to experience.

tion Such a change is not necessarily painful, though spontaneous pain may from this time set in before any actual gangrene occurs Along with the redness or blueness, the skin is apt to be shiny, the forefoot a little swollen, a state of things which the patient's desire to keep on his feet will often exaggerate Moreover, probably because blood most easily reaches a dependent part, the foot is usually more comfortable when dependent, so that the individual must get up at night and hang the leg out of bed Thus a vicious circle of deficient circulation and edema is set up, the edema diminishing the already restricted arterial flow and so adding decidedly to the patient's troubles

By the appearance and behavior of the toes, better than by any other factor, the course of the disease can now be traced Following the onset of painful reddish blueness and edema, some one toe or part of a toe may become purple and then black, the gangrene being of the dry or mummifying sort Very often, as the gangrenous part becomes demarcated and is finally amputated, the appearance of the other toes improves Further attacks sometimes follow, by which other toes are lost, but in the intervals the extremity is of a reasonably good color, the remaining toes limber, free from swelling, and not unduly cold Such a state is shown in Plate II B In other and more serious cases the toes remain cyanotic, cold, and shiny Pain becomes severe It will perhaps have a burning, agonizing quality and with it will go a high degree of hypersensitiveness No one must touch or move the patient's toes, but he will sit in bed gently kneading his foot between his two hands Such a state may last for months, the appearance of the extremity remaining unchanged Dependency gives some relief but increases edema and so works against improvement in the local blood supply Finally, if and when the pain is conquered, the toes are left discolored, stiff and useless, some of the nails so deformed perhaps as to cut into the flesh and threaten local infection Evidently such a patient is hardly better off than if his toes were gangrenous, and indeed his future will actually be made safer if, at a favorable moment, the toes are removed,

**Clinical Manifestations**—Buerger backs up his contention that an inflammatory reaction ushers in the disease by calling attention to certain premonitory symptoms before the peripheral arteries give evidence of obstruction. Such are, "lancinating pains in the legs, especially in the calf and foot, cramp like pains in the leg, first interfering with walking, later requiring complete rest, tenderness in the calf and along the anterior tibial region, simultaneously with, preceded by, or unassociated with attacks of migrating phlebitis." Such observations must be unusual and represent a very acute form of the disease.

*Intermittent claudication* is usually the initial symptom. Its pathologic basis has already been described (Chapter I). Clinically, the pain complained of may be a sort of numbness, or numbness followed by cramp, or pure cramp or even a feeling as if a knife were being thrust into the leg. And it may be felt in the mid calf or in the anteroexternal group of muscles of the shin or even the foot. Sometimes the pain, at its height, ends by moving into the back of the upper thigh and buttock as if the sciatic nerve were affected. But always about the same amount of exercise brings on the pain or disagreeable sensation—walking so many blocks at such a speed. The more fully the pain has been allowed to develop the longer will be the period of rest required for its relief. Walking on a brick side walk brings on the limp more quickly than on the grass. It is more troublesome in cold weather than hot. When the pain is on, the leg is a little sensitive to touch, but not obviously changed. A limp is likely to appear suddenly. Very rarely, in its early stages, it may come and go, in which case a vaso motor element may enter into it, or perhaps a sudden alteration in blood pressure. But as a rule, an intermittent limp, once present, has come to stay.

After months, or it may be years, thickened skin often appears under the toe nails, especially upon the great toe, the nail itself taking on perhaps an unnatural transverse curve. At this time some of the toes, again the great toe in particular, are apt to appear red or reddish blue in the dependent posi-

morning, these toes would turn purple but after walking for a while their normal color would return and the burning discomfort would be for the time relieved. In the next two months, the toes, with the exception of the middle one, improved. Thus, however, grew more discolored, and "there is a burning feeling and also a pressure feeling as if the toe were being crushed" A trophic disturbance in the form of excessive peeling of the skin from the other toes was evident. The left foot sweated excessively. Hot water upon the skin was intolerable. Pain was so severe at night that sleep could only be secured when the left leg was dependent, so that the patient had come to sleeping in a chair. Occasionally, he noticed a cold, stiff feeling in the finger tips of both hands.

Examination showed a young man of sanguine complexion, evidently suffering. No pulsations could be made out in the peripheral vessels of the left foot. The skin of the toes sweated constantly. It peeled freely from the great and little toes (fungus infection?). The color of all the toes but the middle one was normal enough but all were hyperesthetic. The patient feared to have them touched. The left middle toe was purple, its outer portion black, dry, and shrunken.

Amputation of this partly gangrenous toe through the metatarsophalangeal joint left a dry reactionless wound which failed to heal. Meanwhile the burning pain grew worse and other toes began to show patches of gangrene, notably the great and second.

A month after the local amputation, the patient's state was pitiable. Pain was continuous. The amputation wound, though very little infected, was unhealed. The outer half of the great and second toes was now gangrenous. On the ground that this was Raynaud's disease, some observations of reactive hyperemia in response to the application of an Esmarch bandage to the thigh were made but were not followed up. The foot flushed slowly, the toes not at all.

Amputation was performed six inches below the tibial tubercle, a closed procedure which entirely relieved pain and which healed ideally. Pathological examination showed that the pos-



a prophylactic sort of amputation occasionally used by those very familiar with the disease

The gangrene of thrombo angutis obliterans, when it occurs in such a foot as has just been described, starts in a small way, beside a toe-nail, beneath a callus, in the outer half of a toe. Indeed, it resembles the arteriosclerotic sort, except that there is apt to be more cyanosis and swelling of the other toes and near by foot. Above all there will usually be far more pain and that of the characteristic agonizing sort. In the most serious cases, more than one toe, even a part of the foot is apt to become necrotic. Pain and hypersensitiveness are sometimes relieved by amputation of the gangrenous toe or toes but more often are not. Under these circumstances, the wound of a local amputation often fails to heal, becomes the starting point for more local infection and leads to an extension of gangrene. Such is the worst form of the disease, the sort which so often ends in amputation of the limb.

The following are cases illustrating respectively a very acute, malignant form of the disease, a serious sort, yet more amenable to treatment, and a variety so tractable that it can almost be considered to have been cured. Smoking is an obvious factor in all three. The cases were not treated upon any consistent plan. They are selected because a good deal of positive information about them has been secured, permitting the clinical signs and pathologic background to be compared.

W J D, twenty one years of age, a Massachusetts born Irish American, came under observation in 1916, complaining of a sore toe. He was a vigorous young fellow who admitted smoking only fifteen to twenty cigarettes a day. The patient's race, the date, and the smoking are emphasized because at that time it was hardly believed that thrombo angutis obliterans could occur in any but Hebrews or that the cigarette could be so serious an aggravating factor as it is now believed to be. The patient continued to smoke throughout his illness.

For a vague period, both feet had felt cold, the left more than the right. For five months, a burning sensation had been noticed in all the toes of the left foot. On first standing in the

About the next case, distinctly less acute, much information was secured by surgical methods which should be condemned for general use, and which were here more successful than they deserved to be

W J S, a Massachusetts born man, twenty eight years of age, without Jewish blood but a cigarette and pipe smoker, had suffered for about eight months before coming under observation from "tightness and pain in the muscles of the left calf on walking" His occupation, in a shoe factory, had previously caused him to stand for long hours and during the three previous years he had suffered at irregular intervals from attacks of swelling and pain in the entire left leg Through these attacks, which were self limited, he worked Their exact nature can hardly be surmised His intermittent limp was such that he could walk no more than a hundred yards without bringing on the cramp like pain in his calf There was no radiation For three months the region of the great toe nail, especially its outer half, had been dark blue in color The cyanosis faded out upon the base of the great toe and the adjacent foot

Examination showed a strong, stocky fellow, suffering from only a very moderate spontaneous discomfort in his left great toe The cyanosis has already been described. It was faintly yet unmistakably present on the toes of the right foot as well The left foot was a little cooler than the right Investigation with the thermocouple (in the early days of its use) gave very similar temperatures for both feet but there was a very abrupt change just above the left ankle from warm (above) to cold (below) The dorsalis pedis pulsation was absent on the left, faintly present on the right

Exploration of the left posterior tibial artery behind the internal malleolus, a procedure which might well have resulted in a gangrenous wound, disclosed a shrunken obliterated artery in the midst of a plexus of dilated veins The wound was made and closed with minute care The patient was very fortunate indeed that it healed without reaction

Exploration of the left femoral artery in Scarpa's triangle

terior tibial artery had been obliterated by thrombosis. The stump supported an artificial leg for fourteen months. At the end of this time a vesicle formed upon its end and though soon healed, gave warning of what was to come, namely, intractable ulceration.

Some two years and a half after first coming under observation, the left leg was amputated through the lower thigh. Again healing occurred, again pain was relieved. Thrombosis had now occupied the anterior tibial vessels.

Six months later, the patient was using crutches, walking on his right leg which had begun to exhibit the disease in the form of a bursting feeling in the right second toe. However, the Esmarch bandage was now used intensively to secure a reactive hyperemia. The flushing time would grow shorter, then longer, but pain was gradually relieved. Three years later the right leg was, if anything, better, yet the disease had begun to affect the hands. The fingers were numb and cold, especially the little fingers. At this time the patient drifted to Mexico and has since been lost to sight.

After the first violent attack, the disease in this case seemed to strike rather less strongly, yet its progress, though slower, was never altogether stopped. It was peripheral in type, accessions of gangrene coinciding with outbreaks of thrombosis and obliteration of the chief peripheral arteries. It would be interesting to know if the patient ever gave up tobacco. Doubtless the application of reactive hyperemia saved, for the time being at least, the right leg. This is in accordance with general experience, that if an individual can be carried through a bad attack of painful threatened gangrene without a resort to amputation, the collateral circulation which he subsequently establishes by postural exercises and other routine measures will usually save his leg. The toes, however, in such a case as this will usually have been left so cold and badly nourished—their nails deformed, their joints stiff—that they are not only useless but obviously threaten further gangrene and sepsis. As already explained, their removal, if it can safely be performed, is desirable.

in the dorsum of the foot, began to trouble him when he used his clothes pressing machine. It was this which drove him to seek advice. There was no spontaneous pain.

The patient was a middle sized, slender man who presented nothing abnormal except for his feet. The blood pressure was 130 mm systolic. The distal half of the left great toe was deep blue in color, the rest rather less cyanotic but more so than the right toes, which were only slightly discolored. A very distinct moisture of the left foot was taken to be a sign of sympathetic irritation. No pulsations could be detected below the femoral in either leg. Upon cutting off the circulation for five minutes by means of an Esmarch bandage, the reactionary flush advanced rapidly to the base of the toes which did not become colored for twenty to thirty seconds.

Since the signs of disease were of unusually brief duration, the skin in good condition and the patient comparatively young, it was decided to explore the various accessible vessels. The left posterior tibial artery was exposed at the ankle. It was not thrombosed and pulsated very faintly.

The left popliteal artery was exposed by a transverse incision. Here again there was a very feeble arterial pulsation as if the artery were thrombosed a short distance above. The current might of course have been retrograde.

Both these wounds healed well.

Some days later, the left common femoral artery was exposed at the bifurcation of profunda and superficial femoral. The latter was obviously much thickened, as if distended by an embolus, so much so as to be decidedly larger than the common femoral. No pulsation could be detected in the thickened vessel but some blood was evidently passing through it, for, upon being opened, it was found to have the appearance of a rubber sponge. That is, the thrombus which had recently occluded it had been partly organized and canalized. Behind it could be seen several great, soft pulsating branches of the profunda.

Inasmuch as the canalized vessel seemed incapable of carrying a good volume of blood and because a better collateral

was now made The artery proved to be large, soft and very actively pulsating It was not disturbed

Finally, the popliteal artery was exposed It was small, rather thick-walled and, though a feeble current evidently passed through it, did not pulsate A fine bougie passed up into the femoral met an obstruction half way up the thigh Evidently the artery was thrombosed at this point On the principle that the current through the popliteal was feeble and that a better collateral circulation would form if it were divided, a short stretch of it was resected It proved to have been thrombosed and canalized

The result, as is so often the case in arterial resections—corresponding with the experiences of Dean Lewis and Reichert—was favorable The foot became free from discomfort, its color improved and when the patient was dismissed a few weeks afterwards, he was greatly encouraged Two years later, after moving to California, he reported that his improvement had continued, yet his intermittent limp remained Indeed his left became his best extremity, for his right leg was amputated, first below, and later at the knee, and his fingers gave him some trouble Probably he never gave up tobacco Curiously enough, coffee seemed to affect his circulation unfavorably

This case demonstrates, what can now be learned by arteriography, that a good sized artery once thrombosed and canalized is so narrowed as to be incapable of transmitting an effective stream Evidently here the thrombosis occupied both the medium sized arteries of the leg and the superficial femoral

The last case to be quoted is an example of a very mild disease which might have done very well under abstinence from tobacco, which the patient actually gave up, and the postural exercises which were used However, exploration of his vessels in search of information was not only very enlightening but apparently contributed to the rather dramatic result

S B, a Russian born Jew, forty years of age, a cigarette smoker from the tender age of nine, presented himself because of a typical left sided intermittent limp The pain brought on by walking was knife like After two months, a second pain,

Vasomotor signs take the form of unnatural changes in color in response to a cool environment and to emotional upsets. Sweating, which occurs in the abnormal foot while the other is dry, is evidence of an associated sudomotor excitement. A toe which turns blue, then white, then pink again has gone through a cycle of nearly arrested circulation and recovery suggesting vasomotor irritation. And if an individual, subject to vasospasm, is tested by first raising and then lowering his leg, as described in the opening chapter, it will be found that the tips of some toes are very slow indeed to color, remaining white long after the rest of the foot is pink. Yet if a reactionary hyperemia is excited by any of the customary methods, the tips of those same toes will then color like the rest. Vasospasm is likely to be suspected when blueness (or pallor) and coldness of a foot is associated with the presence of a fair arterial pulsation in the dorsalis pedis artery. It should be looked for in excessive cigarette smokers, especially when, with no threat of gangrene, a sudden change to a cold skin temperature is recognized as the hand passes over the ankle toward the foot.

The final and authoritative test of sympathetic irritability is of course paravertebral blocking of the sympathetic chain with procaine, or, if it is unnecessary to compare the state of two legs, spinal anesthesia. A posterior tibial block with procaine will, if successful, abolish arterial spasm and bring forth a rise of temperature in the toes and sole. A positive is more significant than a negative test. For though the latter suggests an organic constriction, the result may really be due to failure of the procaine to penetrate the nerve.

Vasomotor spasm is important to recognize because it is so often relievable by abstinence from smoking, by avoiding cold and emotional stress, and especially by the various forms of treatment presently to be described.

**Diagnosis**—In the previous chapters, the differential diagnosis between arteriosclerotic vascular deficiency and that of thrombo angitis obliterans has been discussed. There the following points were emphasized as compared with arterio

circulation would be established if the artery were divided, a block eight cm long was resected

Immediately the toes of the left foot became warmer than the right (lessening of reflex sympathetic irritability) and within a few days had taken on a normal color. In four weeks, under routine treatment by passive vascular exercises and withdrawal of tobacco, the threatened gangrene of the left great toe had cleared up. After this, except for the fact that a year later an area of phlebitis migrans was noticed in the left popliteal region, the disease continued to recede. Four years later, the patient could walk and even run without a limp. The toes of *both* feet continued to be of good color.

It is not contended that cases like this, obviously favorably affected by leaving off smoking and the institution of vascular exercises, should be treated by resection of the femoral artery or even exploration of the peripheral vessels. The case is put forward as a central, as opposed to peripheral, type of disease, that is, in respect to the limb itself, and to illustrate the vasodilating effect of resecting a thrombosed artery, a cause of peripheral sympathetic irritation and vasospasm. It will be noticed that under routine treatment the early signs of thrombo-angitis obliterans in the opposite leg receded.

**Vasomotor Manifestations**—It has been suggested above that vasospasm is sometimes an element in the ischemia of thrombo-angitis obliterans and may arise under either of two sets of circumstances: first, because such vasomotor nerves as happen to be associated with a stretch of inflamed artery and vein are engulfed in the inflammatory process and, being irritated, excite a vasospasm within the sphere of their influence, both in very small vessels, as in the toes, or in such large vessels as the femoral, and second, because inflammation of a large artery sets up, directly or reflexly, a chronic vasospasm in the vascular tree peripheral to it. That this second type of vascular reaction is a real one, however obscure its mechanism, is vouched for by the really dramatic change in the peripheral circulation which often follows resection of a stretch of inflamed thrombosed artery (and perhaps vein).

intermittent limp, in one leg or the other, of coldness in the feet, of difficulty in getting the feet warm at night Perhaps the great toe and some part of the forefoot on the affected side will tend to become cyanotic and painful on long standing Pulsation in the arteries of the affected foot will very likely be absent, in the opposite foot, feeble It may be taken for granted that thrombosis has occurred in the larger arteries below the knee, or even in the femoral above The prime object of treatment is to encourage the establishment of a collateral circulation

*Smoking* must utterly be abandoned Whether the matter, as Collier and Maddock have shown, is purely one of temporary vasoconstriction with each cigarette or whether tobacco exerts an allergic influence is immaterial It is agreed today that tobacco smoking, and particularly the inhalation of the cigarette, seriously aggravates the disease and stands in the way of the development of the collateral circulation, the vital widening of the vascular bed Cutting down the number of smokes is useless and keeps the habitual smoker irritated and dissatisfied The individual should know that he will never smoke again It will presently be suggested that the pain of impending or actual ulceration or gangrene is more favorably affected by abstinence from smoking than by any other influence But merely because at an earlier stage of the disease the effect of tobacco is less obvious, is no reason for not giving it up

*Vascular Exercise*—The influence which causes a collateral circulation to increase is, fundamentally, reactionary hyperemia Just how a generalized vasodilatation of the smaller arteries of a limb shall be secured is immaterial It is not even certain that for every individual there is one best way To begin with, vasoconstriction must be avoided Smoking has already been discussed. Exposure to cold is nearly as harmful Not only must the feet be protected during the day in cold weather by woolen socks and thick dry shoes but they must be warmed at night by the warm water bottle and bed socks And since cooling the body or even one extremity causes vasoconstriction of the hands and feet, it is about as important to



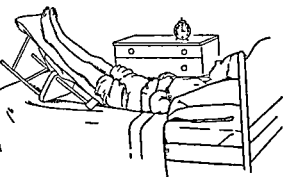
sclerotic disease, thrombo angutis occurs in youngish individuals between the ages of twenty and forty years, and almost exclusively in males, it is ushered in by intermittent limp, the characteristic reddish blue discoloration of the toes appearing considerably later, it is more given to spontaneous pain and that of a severer character, especially after gangrene has set in, its gangrene is far less extensive, it is sometimes associated with migrating phlebitis, and occasionally it shows itself in the upper limbs

In distinguishing thrombo angutis obliterans from Raynaud's disease and from the various states of chronic vasomotor spasm, the above criteria are equally useful. In none of the states of pure spasm, temporary or permanent, is there intermittent limp, nor is there involvement of both lower limbs in young males. Raynaud's disease is a rare disease almost exclusively of females, which shows itself predominantly in the hands. The vasomotor changes in thrombo angutis obliterans unmistakably overlie a disease of chronic vascular deficiency. By tests of skin temperature, it will be found that the toes cannot by any sort of reactive hyperemia be warmed to a normal level. Indeed, it will seldom be possible to raise their temperature more than a very few degrees as compared with the internal temperature of the body.

### TREATMENT

The various stages and forms of the disease require particular treatment, and even those most experienced in its management are not agreed as to the value of certain procedures. It is proposed, therefore, to describe, first, the generally accepted method of treating the pre-gangrenous stage, second, the general plan of treating the disease when gangrene is actually present, and, finally, a number of special forms of treatment which in various hands have proved useful but for which the indications are by no means clear.

**Treatment of the Pre-gangrenous Stage**—It is hardly correct to speak of such a stage since gangrene need never develop. The patient will probably have complained of an



### Position 1

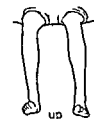
About  
two minutes  $\pm$

Feet to be  
fully blanched



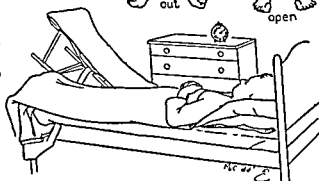
### Position 2

Exercises for  
one to three  
minutes



### Position 3

About  
five minutes



Each series to be done three times

Four sessions daily

FIGURE 6 BUEGER ALLEN EXERCISES

protect one part as another By avoiding vasoconstriction, the stage is set for vasodilatation

The Buerger-Allen exercises, carried out as a ritual, are superior to any other measure which does not involve the use of a complicated apparatus The legs are placed successively in three positions

*Position 1* Lying on his back, watch in sight, the patient rests his legs upon an inclined plane raised to an angle of  $30^{\circ}$ – $45^{\circ}$  He keeps them so raised until the feet are thoroughly blanched, a matter requiring, as a rule, two minutes

*Position 2* The legs are lowered and the patient sits with the legs hanging over the edge of the bed, at least, this is the usual direction, but as the side of many beds is higher than the middle and actually offers something like a ridge which presses into the back of the thigh, partly cutting off the circulation, a better position is secured by resting the buttocks against the edge of the bed and letting the legs sprawl out relaxed, the heels resting on the floor However that may be, while the legs hang dependent, the feet and toes are put through a series of motions the ankle is flexed *downward*, then *upward*, the foot is rocked *inward* (tibial flexed) then *outward* (fibular flexed), the toes are *spread* (extended), then *closed* (flexed) As these exercises go on, the feet are becoming flushed They should turn a strong pink, well out upon the tips of the toes, a matter requiring one to three minutes But if they become cyanotic or painful, they should at once be elevated

*Position 3* For five minutes the patient lies supine, the legs horizontal in bed and wrapped in a woolen blanket warmed by a hot water bottle or electric pad In this way the reactionary flush, secured by position two, is maintained

The cycle is put through three to six times at one session

The sessions are repeated two to four times each day

A favorable effect is marked by a quicker and more complete flush on depressing the toes after elevation, by a better color of the feet, by an increased range of walking (without exciting the limp) and, rarely, by the return of an absent pulse in the

supply sufficient blood This is the defect of exposure to dry air overheated under a cradle

The sitz bath, used at a temperature which feels comfortably hot to the patient is free from danger and usually produces a satisfactory hyperemia—as demonstrated by flushing of the skin The legs and thighs, that is, the lower half of the body should alone be immersed A period of ten minutes is sufficient The sitz bath can be used to start or end the day, even at both times It comes best, perhaps, after the last exercises of the day, just before going to bed, especially if the patient suffers at all from spontaneous pain

Diathermy, where suitable apparatus is available, especially when the individual is confined to bed and suffers spontaneous pain, is recommended by some If it actually conveys heat to the deeper parts, it should do as much as any other agency is capable of doing, and there may perhaps be some advantage in directing heat to some specific locality However, it is ordinarily sufficient, with a large machine, to place each foot upon a metal electrode in order to route the current through both legs With a small machine, diathermy can be applied to one leg or a part of one The amount of current must be gauged by the patient's reaction, his sensation of warmth and comfort For ambulatory patients treatments every other day may be sufficient Bed patients can be treated daily

*Vascular Exercise by Special Methods* more elaborate than the Buerger Allen exercises include (1) the Oscillating Bed, (2) the Suction and Pressure Boot and (3) Intermittent Venous Occlusion

The Oscillating Bed is an expensive labor saving device for doing Buerger's exercises The patient need make no exertion and the exercises can be continued day and night Whether the use of the bed entails any disadvantages, as for instance in the direction of a bad effect on the thoracic organs or brain, does not seem to be known It certainly is not an essential means of treatment

The Suction and Pressure Boot has not been particularly useful in the treatment of thrombo angitis obliterans Not

dorsalis pedis artery Such spontaneous pain as may be present is apt to be relieved Except for the favorable reaction to abstinence from smoking, which is apt to be rapid, the improvement is usually gradual So far as active exercise is concerned, walking should not be attempted except within the limits of comfort, that is, the intermittent limp should not be excited The value of exercise lies of course in overcoming muscular atrophy and tends to break up the vicious circle of atrophy and a diminished vascularity of the muscles

A judicious mixing of rest and moderate regular outdoor exercise should tend to raise the low blood pressure which a considerable number of cases present Raised pressure forces blood more abundantly through narrowed vessels and opens up fresh collateral channels Drugs, except in so far as they may improve the appetite, diminish fatigue, or confer a sense of well being, are of no advantage

In addition, the careful toilet of the toe nails, toes, and feet, as prescribed for arteriosclerotic and diabetic states, should be put into effect the feet and toes dried with a soft towel after the morning wash with warm water, light greasing with a cold cream or lanolin, softening and filing of deformed nails, exquisite care of corns and calluses, and, finally, treatment of any fungus infection which may be proved to be or may even only be suspected of being present

It goes without saying that the blood should be studied for any gross abnormality—no consistent chemical changes encouraging thrombosis have been discovered—and that any such should be corrected, that the fluids should be kept up by an abundant intake of water, and that any discoverable infectious foci should, on general principles, be abolished

*The Application of Heat*—Whether heat should be applied depends somewhat upon the patient's reaction to the routine already described, for the reactive hyperemia and the avoidance of vasoconstriction can of course be secured without recourse to specific means of heating the limbs It has already been explained that too high a degree of heat sets going a metabolic activity for which the circulation is often unable to

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The *Suction and Pressure Boot* has not been particularly useful in the treatment of thrombo angitis obliterans Not

uncommonly, even in the production of negative pressures of sixty to eighty mm of mercury, the heavy rubber cuff which surrounds the thigh seems actually to check the arterial inflow. This is particularly true in the advanced case. In mild cases a reactive hyperemia and a collateral circulation can be brought out by less elaborate and expensive methods. In the presence of gangrene, the use of the boot leads to a spread of infection. However, it will be worth while to try the apparatus on many cases in which spontaneous pain without gangrene is a feature. In unpredictable instances, pain is certainly relieved. It will *not* be worth while to make financial sacrifices to secure the use of an apparatus which can accomplish little not equally obtainable by simpler means.

*Intermittent Venous Occlusion*—Any one possessing a blood pressure machine can carry out this treatment, though for routine hospital use the apparatus of Collens and Wilensky is labor saving and capable of repeating the occlusion with whatever pressure is desired. For persons with low blood pressure and a deficient peripheral circulation, the compression, which in any case should not be higher than the diastolic arterial pressure, should range perhaps from forty to sixty mm of mercury and should not be maintained for more than two minutes at a time. A broad cuff for the thigh is desirable. De Takats has pointed out the advantage of elevating the leg for a minute or so after each compression—to drain it of blood—and the disadvantage of continuing the exercise for bouts of longer than one half hour.

Intermittent venous occlusion should prove more useful in the treatment of thrombo angitis obliterans than suction and pressure in the boot, for, as already explained, it produces pretty much the same physiological effects but without unduly compressing the feeble arterial stream. It certainly requires less expensive apparatus and is easier to adapt to home use.

*Intravenous Saline Injections*—It is very difficult to know what to say about this particular treatment. The suggestion of diminishing the blood's viscosity came from Japan. Willy Meyer introduced it into this country, and such experienced

chancians and investigators as Samuels and Silbert swear by it. Originally, a physiological salt solution was used, sodium citrate solution has been tried, and after considerable experience with hypertonic saline at a strength of five per cent, the tendency at present is to use a two or three per cent solution of sodium chloride.

There is no question that at one time rubber tubing and salt solution insufficiently freed from foreign material—in other words, dirt—had a good deal to do with the favorable effects the patients experienced or thought they experienced. They were, in fact, subjected to protein shock with its accompanying chills and fever. Doubtless this aided vasodilatation. Yet it seems to be established, by oscillographic tracings as well as by clinical experience, that the repeated intravenous injection of slightly hypertonic clean salt solution enlarges the amplitude of the peripheral pulse. The effect of any one injection may last several hours. The patient is said to notice first a feeling of increased warmth in the affected leg. Trophic disorders tend to clear up. Above all, the intermittent lump is said to improve so that the patient can walk farther and faster. A decided advantage of the treatment is that it can be used in the presence of gangrene and ulceration when passive exercises and intermittent suction or venous compression may be harmful. Also it offers the patient a routine whose potential usefulness he can see and feel, an important consideration as those will testify who have had to see patients through a long bout of gangrene and agonizing pain. Its disadvantage is the nuisance of repeated venipuncture and the preparation of the salt solution and tubing, a matter of great difficulty outside of well equipped hospitals.

According to Samuels, in preparing the three per cent solution, nine grams of chemically pure sodium chloride are added to 350 ccm of doubly distilled water. After filtering into a 500 ccm pyrex glass flask, the solution is either sterilized in a pressure autoclave for ten minutes or boiled vigorously for the same time. The extra fifty ccm are to allow for evaporation. For a two per cent solution six grams of salt are used.



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*Intravenous Saline Injections*—It is very difficult to know what to say about this particular treatment. The suggestion of diminishing the blood's viscosity came from Japan. Willy Meyer introduced it into this country, and such experienced

The surroundings should be as cheerful and diverting as possible. An abundant fluid intake should be maintained. The leg should be kept warm by woolen coverings. If the toes are not actually gangrenous or ulcerated, Buerger Allen exercises may be used, or intermittent venous occlusion may be tried, or alternating suction and pressure in the boot. Heat can be applied in the form of diathermy, or a properly controlled electric pad (if there is such a thing) outside the woolen wrappings. In other words, if ulceration and gangrene are absent, all means of exciting a reactive hyperemia will be tried, with the expectation that pain, if present, will be relieved as the peripheral circulation is improved.

In the presence of ulceration and gangrene, vascular exercise will have to be confined to intermittent venous occlusion (arterial occlusion may cause additional thrombosis and though useful if successful, is a dangerous gamble) and even the occluding venous pressure must be light—hardly more than forty mm. of mercury. Under these conditions, a trial of two or three per cent saline solution, to be injected in an amount of 300 ccm. three times a week is certainly worth considering. Diathermy can still perhaps be used. To assist sleep and to assuage pain, alcoholic drinks will be of some help as well as offering aid in vasodilatation. For drugs, the barbiturates will have to be used, morphine never, though codeine sulphate may perhaps be combined with the other sedatives.

The local treatment of an ulcerated area, as for instance the common form beside the margin of a nail, may properly consist in a daily short soak in a warm solution of almost any mildly antiseptic sort. This is to loosen adherent secretion and favor drainage. A freshly prepared solution of chloramine, 1-200 is satisfactory. The watery solution of iodine in the form of Lugol's solution 400 times diluted is not irritating. A watery solution of a cocoanut oil derivative, anything clean will do, including a saturated boracic solution or normal saline provided it is sterile. Then the sore should gently be dried with cotton swabs, the adjacent skin cleaned with cotton and a neutral soap, wiped off with the same antiseptic already

The original solution is resterilized eight hours later for an other ten minute period. When a needle of nineteen gauge is used, the injection should take about ten minutes. Three injections a week are advised, for a three months' period, and are then gradually discontinued during the next six months. For patients whose oscillometric index is zero or less than 0.5 at the ankle, a longer period of treatment is recommended.

*Diet and Drugs*—A diet high in calcium and low in potassium has been suggested, apparently because of its favorable affect on vasospasm. Perhaps it is more important merely to see that the patient has an abundant, *attractive*, varied diet, with the idea of keeping up his nutrition during the difficult stage of the disease. Three good meals a day are something to look forward to during an otherwise boring if not actually painful few weeks or months in bed. Vasodilating drugs can not be recommended.

Sedatives and hypnotics are a problem. Opium and its derivatives are absolutely barred. If effective, they set up a habit, but actually they soon lose their effect. The routine measures already described must be relied upon to subdue pain, but for sleep the barbiturates are probably the most useful. From the great number of preparations available some few will usually be found to be satisfactory.

**The Treatment of Threatened or Actual Gangrene**—Patients showing deep cyanosis in one or more toes, or ulceration, or actual gangrene, usually suffer also from spontaneous pain. The vicious circle of edema, a defective arterial supply and a preference for the dependent position (to relieve pain) is therefore almost certain to be present. Smoking is first of all barred. The patient is confined to bed and if possible to a bed whose parts can be tilted so that the foot can be raised for a time or lowered. (This does not refer to the automatically tilting beds earlier mentioned.) The plan is to keep the affected leg at least horizontal for nearly the whole twenty four hours if possible—to get rid of the edema—and only when the patient can bear elevation no longer to secure relief by the least possible amount of depression.

He may even employ paravertebral sympathetic resection. The indications for all such special procedures are not clear. Only a considerable familiarity with the ins and outs of thrombo-angitis obliterans and of the procedures themselves justify their use on special occasions.

### SURGICAL TREATMENT

**Peripheral Nerve Section**—Though frowned upon by many and requiring a high degree of technical skill and judgment, section of the sensory nerves supplying the sole and toes may offer the only alternative to a major amputation in the presence of uncontrollably painful gangrene. Actually, since the importance of cigarette smoking has been recognized and abstinence from tobacco has been enforced, the very painful gangrenes are seen far less often than formerly. It may be possible, therefore, to carry the individuals suffering from the more painful types of disease through their bad weeks or months without resorting to nerve section. Two procedures are available: (1) blocking the sensory branches of the superficial and deep peroneal nerves just below the head of the fibula and (2) blocking the superficial peroneal, anterior tibial and posterior tibial nerves in the lower middle third of the leg.

*Blocking the superficial and deep peroneal nerves at a high level*—This procedure is done through a three- to four inch incision which begins just above and medial to the head of the fibula and is carried straight downwards. If carefully handled, the wound is almost certain to heal. The common peroneal nerve is found dividing in the substance of the extensor digitorum longus muscle, which is extensively split. The important motor branches come off within two inches of the head of the fibula. The sensory branches can be identified electrically but this is hardly essential because even if a partial foot drop results from crushing the supposedly sensory branches (as happens in perhaps fifty per cent of the cases) the benefit of the procedure is still greater than its demerits, and as the nerves recover, the moderate degree of paralysis disappears. The sensory nerves are best crushed with a hemostat for per

used, and the area next to the ulcerated or gangrenous surface covered with vaseline gauze. An actually gangrenous part can be treated like an ulcer unless the gangrene is already so dry and the area nearby so clean that anything but a sterile dry dressing would be out of the question.

To aid in controlling pain, an analgesic drug may be added to an antiseptic ointment. Such may be successful if an ulcerated surface is present but can have little or no effect on normal skin or gangrenous tissue. Ethylaminobenzoate ointment, ten per cent, or nupercaine, one per cent in petrolatum, may be useful.

When a large open surface is left, as after the casting off of the end of a toe, the exposure of a joint or the formation of a crevice between the toes, the use of Dakin's solution may prove possible. The full ritual must be used, with protection of the skin by vaseline gauze. The only preparations of the hypochlorites are often preferable, being less painful, as for instance dichloramine T in chlorococaine, or eusol and mineral oil freshly mixed in equal parts. Combinations of zinc oxide, mineral oil and cod liver oil promote healing and epithelization.

Should a good line of demarcation form at the base of a toe, with dry gangrene peripherally and reddened reactive skin proximally, amputation in the zone of reaction is permissible, but such a state as this will seldom present the problem of painful gangrene for which treatment is so difficult. In other words, local amputation, even if surgically possible, will seldom relieve the pain of gangrene. And when, as a result of the casting off of gangrenous tissue and the appearance of healthy granulations, the tissues are ready for healing, the battle is won anyway. In the presence of signs indicating this outcome, conservative treatment is continued, however slow progress may be.

In aid of such treatment as the above, the resourceful surgeon may invoke the assistance of some of the procedures described in the following paragraphs, as for instance, sensory nerve block, periarterial sympathectomy, or arterial resection.

sympathetic supply to a limb by removal of the ganglionated chain in the lumbar region, or dividing the preganglionic fibers in the upper thoracic, may be counted a last resort in the treatment of thrombo angitis obliterans. If the routine measures—abstinence from tobacco, rest, protection from cold, a liberal diet, and passive vascular exercises—fail to cause improvement, the procedure may be considered. But before accepting the operation, one must go much further. If results of any consequence are to be expected, it must be shown, by the trial of some of the tests of reactionary hyperemia described in the opening chapter—brief arterial occlusion, heating the other limbs or the body, the intravenous injection of sodium nitrate or, best of all, spinal or paravertebral sympathetic block—that the peripheral cutaneous temperature can be decidedly raised or the peripheral pulsations increased in the limb in question. Using these criteria, sympathetic resections will not often be performed. However, in individuals reasonably young, as sufferers from this disease must usually be, and in persons whose general health is reasonably good, the operations present no especial difficulty. For the lumbar sympathectomy, an extra peritoneal approach through the flank, leading to removal of the second and third lumbar ganglia, is satisfactory. For the upper thoracic operation, there are two approaches,\* from above the clavicle and from the back. In case of threatened loss of the fingers, it is believed that the upper thoracic resection is usually worth trying. That an alcoholic injection, as described in the previous chapter, will serve the same purpose is hardly to be expected. Yet it may not inflict a new pain and will probably last long enough to encourage the formation of new peripheral vascular channels.

In case of threatened loss of a leg, the lumbar sympathectomy is of doubtful value. If pain, plus gangrene, for which amputation is usually performed, is not relieved by other means, lumbar sympathetic resection is also likely to fail.

As a cure for intermittent limp, lumbar sympathectomy is not recommended.

\* These operations are fully described in the following chapter.

haps 0.5 to 1 cm. Regeneration does not recur until the patient has been tided over the difficult period.

*Blocking the superficial peroneal, anterior tibial, and posterior tibial nerves*—This operation, as devised by Smithwick and White, is performed rather below the middle of the leg but not below a point five inches above the ankle joint. Plate IIIB, page 107, shows the anatomy of the nerves and the distribution of the cutaneous branches. The posterior tibial supplies the sole so that crushing this nerve leaves that part, together with the tips of the toes, anesthetic and paralyzes as well the intrinsic muscles of the foot. The only advantage of the lower incisions is to avoid the possibility of toe drop. The wounds must be made with great care lest sloughing occur. Smithwick and White advise making the incision to reach the anterior tibial midway between the tibia and fibula. The superficial peroneal can be picked up through this same incision. For the posterior tibial, the incision is parallel to the posterior angle of the tibia. The cleavage plane between the flexor digitorum longus and the soleus gastrocnemius muscle is the guide to the nerve. The nerves are to be crushed with the hemostat.

The injection of alcohol into these nerves too often results in an alcoholic neuritis causing pain nearly as bad as the original. But there is no objection to dividing the nerves as Silbert has suggested. Indeed Silbert was the first to attempt the relief of pain, using alcohol but without exposing the individual nerves.

The benefits of blocking the sensory nerves go rather further than the mere relief of pain. The dressings become very much easier, and whereas the forefoot may have been so sensitive that Dakinization could not be used, now the application of Dakin's fluid or other hypochlorites is possible. Thus gangrene and ulceration may rapidly be cleared up, and sources of pain eliminated before the nerves can regenerate. Moreover, the peripheral blood supply may actually be improved (release of vasoconstriction).

*Sympathetic Neurectomy "Sympathectomy"*—Blocking the



**EARLY THROMBO-ANGITIS OBLITERANS** **111** **32** and thirty a heavy smoker  
 Mass Gen Hosp 327844 Early, minor gangrene of several toes probably  
 associated with severe funga infection Local treatment and lumbar sym-  
 pathectomy saved toes **B** **34** and thirty-eight a heavy smoker Univ  
 of Virginia Hospital Local gangrene well demarcated Little toe recently  
 amputated Remaining toes healthy and lumbar sympsectomy good **C** **35**  
 aged twenty-four Mass Gen Hosp 217313 a heavy smoker Intermittent  
 limp for one year Local gangrene beneath left great toe and two weeks  
 earlier healed Right foot sudden agonizing pain four weeks earlier Note  
 shiny dark (red) skin and gangrene of third and great toe Rapid advance  
 of gangrene—amputation through thigh Almost certainly sudden occlusion  
 of a large peripheral artery by thrombosis



**Arterial Resection Periarterial Sympathectomy**—In some respects, these two procedures may be expected to act in a very similar way. Both may break up a vicious circle of local vascular irritation and peripheral vasospasm. An example of the good effect of resecting the femoral artery in an instance of femoral thrombosis has been quoted earlier in this chapter. An extensive femoral perivascular sympathetic extirpation acts perhaps in a somewhat similar, if less radical way (It fails of course to divert the stream from a partly closed artery.) But the indications for either procedure are not well established. Neither should be used by any but those most familiar with the disease.

**Amputation**—The operations employed are those already described in the treatment of arteriosclerotic disease, but the criteria are somewhat different. For most individuals will still desire to be active and, if kept active, will be able to earn their own living. The danger of infection is decidedly less. Therefore if the routine observations and tests show a reasonable vascularity, giving hope that an amputation *below* the knee will result in a useful stump, the amputation through the leg should be tried. There is all the more reason to save the knee joint since a disease severe enough to cause the loss of *one* leg will often involve the other to a similar degree, and one natural knee joint is a tremendous advantage, two artificial knee joints being too great a handicap for any but the most courageous and athletic.

*The amputations through the knee joint\* of the Gritti Stokes or Callander type will of course be most generally useful, and a higher amputation is seldom necessary.*

There is no doubt that amputations today are resorted to much less frequently than before the significance of tobacco was generally accepted. The fearfully painful intractable forms of the disease are now less common and doubtless in the future will be rare. In the treatment of thrombo angitis obliterans, amputation of more than the toes is decidedly a confession of failure.

\* These operations were briefly described in the previous chapter.

## Thrombo angitis Obliterans in Women

The rarity of the disease among women who, until recent years, have not been cigarette smokers, has always seemed somewhat of an argument that smoking is an important factor which at least aggravates thrombo angitis obliterans. In 1932, Horton and Brown, of the Mayo Clinic, were able to collect only ten cases in females, out of some seven hundred instances of the disease which they felt might properly be counted thrombo angitis obliterans. The average age of the women was thirty nine, six of them presented characteristic peripheral lesions, and two had suffered from a migrating phlebitis. Curiously enough only two gave a history of intermittent lump. The disease on the whole seemed milder than that of men. Many do not believe that it exists.

The following case is an example of a severe form of the disease \* observed at the Peter Bent Brigham Hospital.

S. L., a married Polish woman, forty five years of age, the mother of one child, desired to smoke cigarettes so continuously that although hospitalized during November, December, and January, she was kept outdoors for the greater part of each twenty four hours.

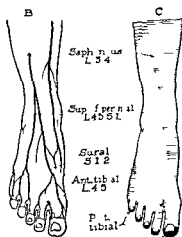
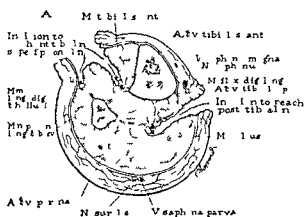
Since the age of fifteen, she had been troubled by an intermittent lump, worse in the right leg than the left. Seven years before first entering the hospital, at the age of thirty seven, she began to suffer from spontaneous pain in the right foot and knee. A white patch appeared upon the right foot which became very cold and as if needles were being applied to it. Rather suddenly, then, the right foot turned purple and the leg was amputated through the right thigh.

While still convalescent from the amputation, the left foot

In a letter some years later the patient recalls her habit as a little girl of eating ergot in the form of the fungus as it grew on the rye. In her own words: "When I was very young I used to walk through rye fields on my way to Church during the month of June just before the rye ripens. \* Some of the kernels would turn black and grow to the size of a small olive. I used to pick those distorted kernels and eat them as they were juicy and acrid tasting and I liked them. Many a time I picked a handful so I ate the poison in its natural state." Possibly her disease is in fact ergot poisoning and not thrombo angitis at all.



1 GANGLIENE OF THE MOST VICIOUS TYPE, IN A FEMALE SL, aged forty five, a very heavy cigarette smoker. Diagnosis of Thrombo angitis Obliterans made upon amputated leg. The other leg had already been amputated several years earlier. History of using ergot.



B DISTRIBUTION OF THE SENSORY NERVE SUPPLY TO THE FOOT. Showing White and Smithwick's method of exposing the various nerves about five inches above the ankle. Crushing them at this level does not cause foot drop, but leaves the sole anesthetic and paralyzes the intrinsic muscles of the foot. (From Homans' *Textbook of Surgery*. Courtesy of C. C. Thomas, Springfield, Ill., and Baltimore, Md.)

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became cold and swelled at the ankle Nevertheless, except for a sense of coldness and "needle pricking" in the left foot, she remained active, using a right artificial limb successfully Heat brought relief of the symptoms of discomfort in the left foot

One month before entering the hospital, the patient noticed, for the first time, a whitish area upon the dorsum of the left great toe In two weeks, this area, which the patient had concealed with a bandage, discharged pus Soon after this, the great toe became gangrenous, the fourth followed suit and an area of gangrene spread across the forefoot At the same time spontaneous pain became fixed and fairly severe Hanging down the foot brought some relief

The state of the foot is sufficiently revealed by the photograph reproduced It will be noticed that the area of gangrene is irregular and that the foot exhibits a moderate edema No pulses could be felt in any vessel below the inguinal ligament

During the next six weeks the gangrene advanced moderately, always having a well marked line of demarcation Pain was rather intermittent, occurring chiefly in a crampy form at night The cutaneous temperatures of the foot and leg were not particularly low There was no difference between that of the thigh and leg No abrupt change anywhere The mouth temperature ranged daily from 99° to 100° F Spinal anesthesia caused no vasodilatation whatever in the left foot The patient could not be prevented from smoking constantly!

Amputation, which, in view of the loss of the other leg above the knee, might well have been attempted below the knee, was made through the lower third of the thigh and healed after only minor sepsis confined to the skin The pathological report was "Thrombo angitis obliterans" and the description of the diseased tissue is consistent with the appearances in authentic cases among men

Two years later the patient expressed herself as being well and cheerful She was successfully navigating a wheel chair and smoking as much as ever

## CHAPTER IV

### SPASM OF THE ARTERIES AND ARTERIAL EMBOLISM

MAURICE RAYNAUD gave the first systematic description of a disease characterized by arterial spasm. As is usual in the observation of a new symptom complex, he included in his account of thirty cases a greater variety than he supposed. It has even been maintained that among the thirty there is only one instance of Raynaud's disease! In which case, posterity would seem to have been rather fussy and ungracious, for Raynaud knew enough about recurrent vasospasm to give a description which has hardly since been bettered. His fault, if any, lay in trying to explain, as a single phenomenon, too great a variety of conditions. Naturally he began with "dead finger." "Madame X", said he, "had been subject since childhood to an infirmity which makes her an object of curiosity to her acquaintances." Any sudden cooling of the atmosphere, even in summer, would cause her fingers to become bloodless, without feeling, and of a whitish yellow color. She would wring them violently or soak them in lukewarm water, whereupon the vascular spasm would gradually relax, to be succeeded by a very painful reaction. That this was a neurosis was suggested by the prompt disappearance of the attacks with the onset of pregnancy.

Raynaud noted the frequency of this sort of thing in young women, especially in times of damp cold, the tendency of such persons to chilblains, in fact, the unnatural reaction to cold, so common in females and by no means unknown in males, in the damp winter climate of western Europe and of England. However, he reasoned correctly that this was only a mild exhibition of a very serious process which could and did lead to nutritional changes in the form of actual necrosis of the

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finger tips, and to a lesser degree, of the toes. It is the easily excited, recurrent, vicious vasospasm, causing minute, oft repeated necrosis with conical deformation of the finger tips, which bears Raynaud's name today, a rare disease. But Raynaud is responsible for very much more than this, for since his time, clinicians have been on the lookout for many other sorts of vasoconstriction—acute, recurrent, and even chronic arterial spasms which discolor fingers and toes, hands and feet—painful states as a rule and very often marked by organic changes, ranging from small necroses and ulcers to gangrene of one or more digits. It has been learned that acute arterial spasm can arise from trauma to a large vessel or even to the limb it supplies, and from injuries to great nerves such as the median or sciatic—causalgias and causalgia like states—resulting in painful ischemia of the peripheral parts, that an arterial embolus not only plugs a great vessel but sets up a widespread vasospasm in the vascular tree beyond, and even that thrombosis in an important vein may excite such spasm in the companion artery as to cause gangrene of an extremity. Still more obscure and peculiar are the vasoconstrictions associated with old infantile paralysis and other nervous disorders.

In the unravelling of these various symptom complexes the names of Sir Thomas Lewis and Rene Leriche stand out, the first for his sound physiological investigations, the latter, for his brilliant reasoning and surgical feats. But many peculiar states remain to be identified and explained, and so the writer makes no apology for presenting the subject of vasospasm with little attempt at order and for including, it may be, in one category conditions which, for all that they are related to spasm of arteries, large or small, may well be due to a considerable variety of causes. After all, the main thing is to recognize in any one case, that vasospasm is or is not the hand behind the disturbance and, having decided in the affirmative, oppose its action as effectively as possible.

At this point it will be well to review the account of the sympathetic system more fully given in the first chapter. Normally, the outflowing sympathetic influences distributed to the

blood vessels of the entire body maintain an even arterial tone. The nerves controlling the muscular arteries are distributed from the spinal cord through preganglionic fibers to the sympathetic ganglionic chain, which lies close beside the vertebral column. Thence, postganglionic neurons pass out with the great nerves of the limbs to ramify at intervals over the larger arteries (Figure 2), and finally, just as sensory nerves are distributed to the skin, so the vasomotor fibers are assigned to the fine arteries and arterioles of corresponding peripheral fields. Thus a general stimulation of the sympathetic system causes vasoconstriction over the entire body, or a stimulus touching only the sympathetic supply to one arm excites vasospasm in that arm. The disorder may even be so local as to affect the vasomotor fibers distributed through a single nerve. Sudomotor and pilomotor excitement is associated with vasoconstriction. A feature of the sympathetic system in the hands and feet is the very rich supply of fine arteriovenous connections capable of being closed or widely opened according as the sympathetic contracts or relaxes their walls. By means of these connections, the surface of those terminal parts conserves heat or gives it out, and thus arterial spasm shows itself more plainly in the hands and feet than elsewhere, particularly in the fingers and toes. The coldness and pallor it occasions are almost necessarily associated with sweating. On the other hand, the arterial dilatation of sympathetic paralysis leaves the skin of the extremities hot, pink, and dry. The variations upon these reactions will be brought out in the account of the various vasospastic states which follows.

#### RAYNAUD'S PHENOMENON

This title is intended to include all recurrent arterial constrictions of the extremities which are excited by cold and the emotions. Such vasospasms are common. It is only the serious, progressive, vicious spasms, which Raynaud himself accused of causing nutritional changes, that are rare. These latter will be described as Raynaud's disease, for that, whatever Ray

naud himself may have written, is the accepted nomenclature of today. With this understanding, the comparatively mild and common form will be distinguished as Intermittent Spasm of the Digital Arteries, a Reaction to Cold. Thus all members of the Raynaud family will be housed under one roof, which, after all, is as it should be, for most of its members appear to be sisters.

**Intermittent Spasm of the Digital Arteries, a Reaction to Cold**—The disease, if so it can be called, starts in childhood or adolescence, rarely later, and is decidedly more common among girls than boys, though there is a familial form, picturesquely described by Hunt in his *Critical Review of the Raynaud Phenomenon*, as "Hereditary Cold Fingers" which appears in both sexes. There are no associated organic peculiarities of the nervous system or in the body at large. The hands rather than the feet are affected, the fingers rather than the thumbs. Symmetry is the rule, even in the unusual event that only one finger exhibits the disease, for instance, the middle finger of each hand may alone be sensitive to cold. The subjects are apt to be thin and, in the countries where the winter cold is damp, are likely to suffer from chilblains. Thus, digital spasm, and for that matter progressive Raynaud's disease, is far more common in England and Europe than America.

The attacks, which often start in childhood, are brought on by exposure to cold, a cold atmosphere, as a rule, during the winter months, but equally well by bathing in cold water. Sometimes, if cold alone has failed to produce an attack, added excitement or embarrassment will do so. There is a great variation in the susceptibility of individuals. Some react to the slightest sort of exposure. Some will only notice whiteness and numbness of a finger after being in swimming for hours, while others will find that all the fingers are involved if they poke their noses outdoors on a cold day. Sometimes the tips of the fingers only are affected. Lewis has called attention to the fact that when the attacks are provoked by placing the hands in cold water, there is a definitely optimal

temperature For instance, water at 15° C (59° F) brings on a spasm, while ice water turns the fingers rather red than white The first sign of local vasoconstriction is pallor of the finger tips The pale area soon turns gray as the remaining blood loses its oxygen, but this initial change may be inconspicuous When the individual first notices her fingers, the tips or even a considerable portion of each have usually become waxy white Actually these changes depend a good deal upon the original color of the skin A rather highly colored skin turns bluish and fingers already pale become waxy Moreover, if the spasm relaxes at moments, fresh blood flows in and soon, giving up its oxygen, makes the skin cyanotic.

The course of any attack will depend upon the length and severity of the exposure If the fingers are made white and bloodless for long, a numbness sets in at the tips and in half an hour may involve the whole of each digit Particularly severe attacks are very disagreeable After a while, as the hands are warmed and spasm is relaxed, a slow wave of bright redness passes out upon the fingers, a reactionary hyperemia, as after any arterial stoppage Under this influence, each finger is apt to tingle, becoming warm and slightly swollen A capillary pulse can often be detected at the finger tip Such a reaction, as Raynaud noted, may be painful

The course of this mild disease is rather favorable than otherwise Rarely it dies out as the years advance Or the individual learns to avoid the combination of events which excites the attack Perhaps the spasm appears so seldom that it can be ignored and repeats itself, rather to the victim's amusement, on occasion Should the attacks become progressively more severe, the disorder must be placed in the class presently to be described as Raynaud's disease

*Treatment*—Madame X did about as well as anyone can. She wrung her hands and placed them in lukewarm water Whether pregnancy relieves others as well as her seems not to be known, so that as a form of treatment it can not be accounted reliable! But of course prevention is really more important There are, as Lewis points out, two elements in

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well to emotional (adrenal) influences. Here the matter may properly be left, with the understanding that the arterial spasm of Raynaud's disease occurs in the digital arteries, the nature of the local fault being obscure. It is only necessary to add that in advanced Raynaud's disease the walls of the digital arteries are actually thickened, so that their capacity is decreased. Under these circumstances, *any* influence which increases vasoconstriction—cold, embarrassment, anger—will be able to close them altogether. Ross has been able to determine the amount of structural change in the digital arteries of persons suffering from Raynaud's disease by the speed and amount of the rise of cutaneous temperature in response to warming the body. In mild, early cases, the arteries are capable of full dilatation. In advanced cases, vasodilatation is very slow and incomplete.

The same sort of individuals—almost always women of youthful years, though the disease occasionally appears in middle age—as are susceptible to the milder form of Raynaud's phenomenon suffer from serious progressive Raynaud's disease. The attacks are now more easily provoked and more prolonged. They start in the finger tips and mount to the base of the fingers, sometimes to the palm, but almost never above the wrist. The first color is usually a bluish one which deepens to slate blue or a dark purple. If the attack is prolonged, the cyanosis is replaced by a waxy pallor. In any case, no return toward a normal color can occur until the hands have been thoroughly warmed. Partial relief is marked by a shift from blue to red as a little arterial blood pushes into the fingers. Indeed, thus betwixt and between state may last for some time, the red and blue areas existing in a patchy way, or perhaps a whole finger will shift back and forth, never really becoming warm and pink. Sooner or later, as the attacks multiply, the pulp of the fingers hardens, the skin tightens and its transverse wrinkles disappear. The fingers taper toward their ends, the overcurved nails projecting beyond the shrunken tips. Upon these tips, little necrotic areas appear in the form of tiny cores of dead skin, which separate

this sort of vasospasm. There is the local spasm which results from direct exposure of the fingers and hand to cold, and there also is the vasoconstriction, in which all the peripheral circulation shares, due to lowering of the body temperature as a whole. Thus the individual must be on the lookout against immersing the hands in cold water, or exposing them without woolen coverings in cold weather, or letting the fingers come in contact with cold metal or glass. But she must also avoid chilling the body or, in case the disease shows itself only in the hands, any other part, especially the feet. It may be well, as is true of those suffering from the serious progressive form of the disease, to begin the day by heating the hands in hot water, establishing a vasodilatation which will not easily react to slight cooling.

**Raynaud's Disease (Raynaud's Phenomenon with Nutritional Changes)** —Lewis's experiments in exciting spasm in the digital arteries by exposure of the fingers, even the base of the fingers alone, to cold, and his demonstration that such spasm cannot be excited by any central sympathetic stimulation, provided the hands are kept warm, strongly indicate that the fault in Raynaud's disease is a local one. If this were all, the whole trouble would seem to lie in the state of the digital arteries themselves. This conclusion, however, cannot unreservedly be accepted. It has been shown that vasospasm in the fingers can take place, especially under the influence of emotion, when the peripheral portion of the sympathetic is absent. Indeed, as Smithwick, Freeman, and White have demonstrated, removal of the ganglia whose cells supply, through the gray, or postganglionic, rami, vasomotor fibers to the peripheral arterial tree, leaves such arteries exposed to adrenal influences, under which peripheral spasm, as excited through the adrenal secretion by any emotion, may be exceptionally severe. Moreover, Smithwick, Telford, and others have found that if they divide the preganglionic rami of persons suffering from Raynaud's disease, leaving the peripheral neurons intact, these persons appear to be cured of their old tendency to peripheral spasm in response to cold and to be resistant as

passed then to a violet tint, then to a slaty white \* \* \* A very cold wind had blown all morning when Rose entered the room. Her cheeks and chin were of indigo colour, her hands were as cold as marble. At the first view I believed them to be gangrenous. The ends of the fingers were of a greenish blue, the palms of the hands were of a deep purple. On the forearms there were marblings similar to those which are present on the legs of persons who use foot warmers. Above the wrists the skin presented its natural colour. These phenomena were less pronounced in the lower limbs, which were clothed with woollen stockings \* \* \* Whilst this young woman spoke, a bright redness began to develop itself at the root of the nose and over the cheeks, then it extended and invaded the blue colour, which soon formed no more than a deep red patch on the tip of the nose, and ended by disappearing entirely. One moment afterwards, the pink colour of the nose began to pale, and this organ resumed little by little its ordinary colour. The same change came into operation at the same time upon the cheeks and the skin, upon the hands the change did not take place so quickly nor in the same way \* \* \* It was at the extremities of the fingers that the cyanosis and the cold persisted longest. Finally at the end of a quarter or half an hour the whole hand was of vermillion red, the pulse had regained its force, the warmth of skin was perfectly developed, and a slight sweat had moistened the cutaneous surface. All these phenomena were reproduced each time that Rose was exposed to cool air, whether in the evening, morning, or at the middle of the day. The reaction only commenced when she returned to her room."

On the whole, the appearance of gangrene away from the extremities and in such parts as the tip of the nose, the cheeks or the pinna of the ear, that is, a juvenile gangrene, is less likely to be caused by a Raynaud like vasospasm than by minute multiple arterial embolism (see page 162). Nor are the contractions of the retinal arteries, which Raynaud observed in one of his later cases, at all characteristic of the typical disease. Vascular spasms, it is now realized, are ex



painfully, leaving minute scars. The process shows no sign of gross ulceration or infection, by contrast with the outspoken gangrene of arteriosclerosis or thrombo angustis obliterans, but is painful, sometimes agonizing. Moreover, so far as the limbs are concerned, it begins in, and is decidedly most advanced in, the fingers rather than the toes. But even if the loss of substance is slight, pain is out of all proportion to it, and healing of the little areas left exposed by the casting off of the tiny necrotic plugs is very slow. However, even in the severest form of the disease, years go by before the fingers are noticeably shortened.

Manifestations of Raynaud's disease in other parts than the extremities are now looked upon with some doubt. However, Raynaud himself, in his second publication and after mature consideration, described them as being a part of the disease. He says: "In the slight cases the ends of the fingers and toes become cold, cyanosed, and rigid, and at the same time more or less painful. In grave cases the area affected by cyanosis extends upwards for several centimeters above the roots of the nails, at the same time the nose and ears may become the seat of analogous phenomena. Finally, if this state is prolonged for a considerable time we see gangrenous points appear on the extremities, the gangrene is always dry, and may occupy the superficial layers of the skin from the extent of a pin's head up to the end of a finger, rarely more." One of his case histories describes changes in the nose and cheeks. This case, Allen and Brown accept but Hunt rejects it because the trouble began a month after what may have been an attack of malaria. However, the account is vivid and decidedly worth quoting.

"Case VI. Rose G., a washerwoman, aged 28 years, with fair skin and bright complexion, enjoyed habitually good health. . . . In the month of March she had several attacks of tertian fever, which disappeared under a sedative. Towards the middle of April she became very impressionable to cold.

"Every time that she went out during weather at all cool, the nose, chin, cheeks, hands, and feet became pale, they

nal secretion. Some of the patients, if exposed, for instance, to the slightest embarrassment, would at once display cold purple fingers. Freeman, Smithwick and White, in two combined researches, showed first that the denervated rabbit's ear was strikingly sensitive to the adrenal secretion and then went on to demonstrate that the arm of an individual deprived of all postganglionic sympathetic supply was equally sensitive. It only remained to find out why the same condition did not obtain, in the case of the leg, following lumbar sympathectomy. The explanation turned out to be anatomical. The customary removal of the second and third lumbar ganglia (the lowest ones receiving sympathetic preganglionic rami from the spinal cord) interrupts all higher sympathetic control of the blood vessels of the leg, but leaves the *postganglionic* rami, emerging from still lower lumbar ganglia, intact. Thus the vessels of the legs are not left without postganglionic control and are not hypersensitive to the adrenal secretion. (See Figure 1.)

This explanation is necessary to account for the success of the procedures which were developed almost simultaneously by Telford in England and Smithwick in America. To free the arm from vascular spasm, all rami passing to the second and third thoracic ganglia are divided and the sympathetic cord is severed below the third ganglion. The rami of the stellate (inferior cervical and first thoracic) ganglion together with the second and third thoracic ganglia themselves are left intact (Figs 7, 8, 9). Time seems already to have proved the correctness of these procedures which differ from each other only in detail and which free the arteries of the arm from any serious vasospasm, whether by exposure to cold or epinephrine injection. Moreover they offer the very decided advantage that if the stellate ganglion (first thoracic and inferior cervical) is left totally undisturbed, both as to its pre and postganglionic rami, the unsightly falling in of the eye (enophthalmos) permanent narrowing of the lids and contraction of the pupil—*Horner's Syndrome*—will not occur. The avoidance of this disfiguring result, with its associated unpleasant sen-

cited by such a variety of stimuli that attempts to group them in a systematic way are as likely as not to increase rather than diminish the difficulty of diagnosis. Spasm of the retinal vessels had better not be regarded as an exhibition of Raynaud's disease.

**Treatment**—The background of treatment is the same as that of the mild and nonprogressive form of Raynaud's phenomenon, that is, avoidance of exposure to cold, cold drafts and contacts, and, to preserve the natural warmth, the wearing of woolen clothing, mittens, and stockings. Ample shoes should be worn in cold weather. The victim of Raynaud's disease will often begin the day by washing the hands in fairly hot water before taking the risk of entering an even cool room. If she can be sure of avoiding exposure afterward and does not fear the habit, she may justifiably take a stiff drink of alcohol before breakfast! Whether physicians should advise such a course is a matter which the writer is not wise enough even to discuss.

*Sympathetic Ganglionectomy* is now so universally if ungrammatically dubbed "Sympathectomy" that only a purist would avoid using the term. Moreover, in the case of the arm, the approved procedure is no longer resection of the ganglionic chain but has gone back to what amounts to a combination of division of the chain and ramisection, as will presently be explained. The story is this. A very satisfactory and complete sympathetic denervation of the arm had been developed by Adson and Brown, following Kuntz's demonstration that the second thoracic ganglion often sent a sympathetic ramus to the lower end of the brachial plexus. They removed, through a posterior approach, the inferior cervical and first and second thoracic ganglia. But the arm, in many cases, only remained free from vascular spasm for a few weeks or months, though the completeness of the sympathectomy was vouched for by the total sudomotor and pilomotor paralysis. It then appeared that the neurectomy was *too* complete, for the degeneration of all the postganglionic fibers left the smooth muscle of the arteries hypersensitive to the adre-

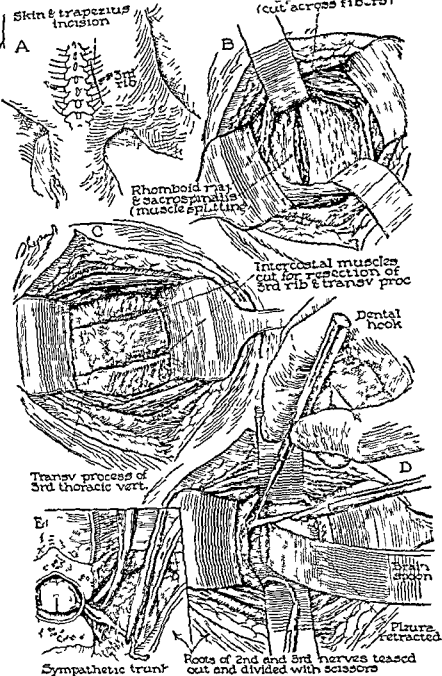


FIGURE 7 UPPER THORACIC SYMPATHECTOMY—POSTERIOR ROUTE. Smithwick's Method. A, B, and C Approach to the second and third left intercostal nerves D Each nerve is cleared of all rami and local branches with a blunt hook and followed into the intervertebral foramen. E Each nerve is gently teased out, exposing the sensory ganglion, and its roots are divided with scissors. The sympathetic chain is cut below the third ganglion and lifted up

sations in the skin of the face and head, is decidedly worth while

Because the operations are now reasonably well standardized and are equally useful in the treatment of the various states of peripheral vasospasm which complicate other vascular diseases of the limbs, they are described below. The indications in Raynaud's disease are that vasospasm should not be otherwise controllable and that the peripheral vessels, those of the digits in particular, should have been proved to relax sufficiently well in response to any effective test of reactive hyperemia (heating the body, paravertebral sympathetic block). That is, it must be possible to raise the skin temperature of the fingers (or toes) to, or nearly to, the high limit and that with reasonable promptness. Otherwise the digital arteries are almost certain to be so fibrosed that the operation will do very little good.

**Sympathetic Ramisection, or Sympathectomy, for the Arm**  
—Two routes are available: the posterior approach, using a modification of the muscle splitting incision advocated by White, Smithwick, Allen, and Mixer, teasing out and dividing the second and third intercostal nerves, in order to eradicate completely their associated preganglionic (white) rami, and dividing the sympathetic cord below the third ganglion, as described by Smithwick, the anterior approach at the root of the neck, using the technique of Gask, dividing the sympathetic cord below the third ganglion, cutting the preganglionic rami to the second and third ganglia, turning up the upper stump and drawing it away from the spine to prevent regeneration, as advocated by Telford.

*The Posterior Approach*, with Smithwick's modification, is here preferred.

An incision is made about eight cm (three inches plus) long, and three cm from the mid line, nearly parallel to the vertebral column (sloping a little upward and outward) and centering upon the level of the second thoracic spinous process (third rib). The fibers of the trapezius muscle are transversely divided. Those of the major rhomboid are split, as are those

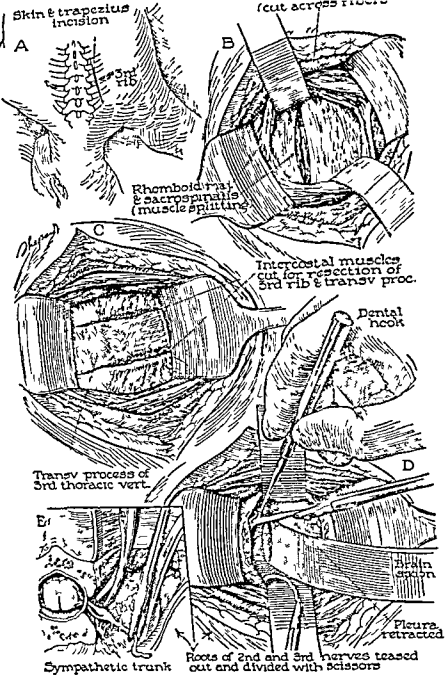


FIGURE 7 UPPER THORACIC SYMPATHECTOMY—POSTERIOR ROUTE Smith with a Method. A B and C Approach to the second and third left intercostal nerves. D Each nerve is cleared of all rami and local branches with a blunt hook and followed into the intervertebral foramen. E Each nerve is gently teased out, exposing the sensory ganglion, and its roots are divided with scissors. The sympathetic chain is cut below the third ganglion and lifted up

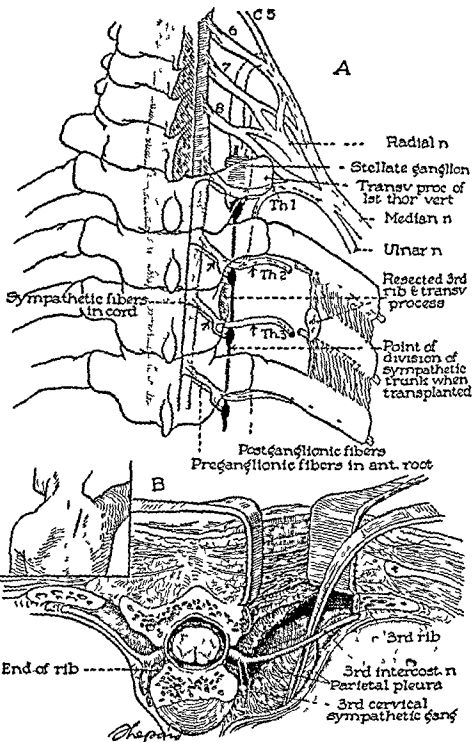


FIGURE 8 UPPER THORACIC SYMPATHECTOMY—POSTERIOR ROUTE **A** Scheme to show plan of preganglionic sympathetic interruption (indicated by arrows) Preganglionic ramus and sympathetic chain in black No pre or post ganglionic rami of the stellate ganglion are disturbed **B** Cross section at level of third left intercostal nerve Operative approach

of the sacrospinalis muscle. The third rib is exposed, isolated, and retracted, its proximal end and the corresponding spinal transverse process being subsequently bitten off with the rongeur.

The pleura is pushed away from the vertebral column with gauze.

A bright, brain spoon retractor serves to reflect light toward the vertebral bodies, or an illuminated retractor is used.

The third intercostal nerve is isolated by blunt dissection, divided at a convenient distance from the vertebrae, followed in to its exit from the vertebral column and freed from all attachments with a dental spatula. In so doing, the posterior root ganglion, with the anterior and posterior roots, are teased out, brought into view and divided. In this way all preganglionic fibers are thoroughly interrupted. A leak of cerebrospinal fluid is unusual.

The second intercostal nerve is similarly treated. In the meanwhile, the sympathetic ganglionated chain has not been disturbed.

The sympathetic is now picked up with a blunt hook between the second and third thoracic ganglia as it lies against the vertebral column and is divided with scissors below the third ganglion. Its upper portion is now turned up, care being taken not to disturb the stellate (first thoracic and inferior cervical ganglia) ganglion. The object of this step is to obviate Horner's syndrome (contracted pupil and enophthalmos) and leave all outgoing postganglionic rami intact. The stump is attached by a silk suture to any convenient fascial structure. The wound is closed in layers with silk. If the pleura is injured, the opening is covered with a fragment of muscle pressed down with gauze.

*The Anterior Approach* (Gask's with Telford's modification) is made through an eight cm (three inch plus) incision, two fingers' breadths above and parallel to the clavicle. A cervical and brachial plexus block plus local infiltration with procaine, rather than a general anesthetic, can be used if desired.



After cutting skin and platysma, the clavicular portion of the exposed sternomastoid muscle is divided and also the little mylohyoid. This step gives access to the anterior surface of the scalenus anticus muscle upon which the phrenic nerve lies. The muscle is cleaned, the phrenic is retracted mesially with a tape and the transverse cervical vessels severed if necessary. After the scalenus anticus has been isolated and divided two cm. above its insertion into the first rib, a view is had of the subclavian artery and the brachial plexus. The latter is severely let alone, not cleared at all. The carotid sheath is retracted mesially.

Before the subclavian artery can be pressed downward, the thyro-cervical axis, which originates from its convexity, must usually be divided between good sized silk ligatures. On depressing the subclavian, Sibson's pleurocostal ligament, which holds the pleural apex against the lower border of the first rib, is revealed. When this has been cut, the pleura falls back and is further peeled away from the costovertebral angle by gentle pressure with gauze.

The wound is now deep and must be lighted by an illuminating retractor or a reflecting spoon. The stellate ganglion is seen just behind the origin of the vertebral artery, between this vessel and the head of the first rib. The chain, below it, is carefully exposed by detaching any loose tissue from its surface. The stellate ganglion must not be disturbed. When the chain has been cut, with long curved scissors, below the third ganglion, the upper stump is lifted upward sufficiently to divide the rami entering the second and third ganglia. It is then attached to any convenient structure with a fine silk stitch. The retractors are now removed and the pleura allowed to fall back.

The only parts requiring suture are the clavicular portion of the sternomastoid muscle—to restore the contour of the neck—and the platysma. The skin is approximated with clips.

Both sides can be operated upon at one sitting provided the operator is sufficiently familiar with the procedure. The patient usually is up and about on the third to fifth day.

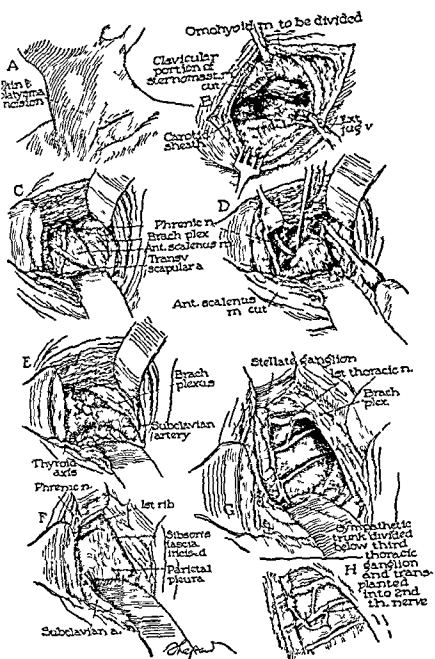


FIGURE 9 UPPER THORACIC SYMPATHECTOMY—ANTERIOR ROUTE. Method of Galt and Telford. A, B, C, D and E, approach. After E, it may be necessary to divide the thyroid axis, in order to depress the subclavian artery. F, Division of pleuro-costal ligament and depression of pleura. G and H, All rami of second and third ganglia are divided, sympathetic is cut below the third and planted into second thoracic nerve (De Takats).

**Lumbar Sympathetic Ganglionectomy**—Though this procedure can be carried out through either a transperitoneal (abdominal) or retroperitoneal approach, the former is never considered at present unless a bilateral operation is contemplated and there is some other reason for opening the abdomen. The transperitoneal procedure is not only much more disturbing to the patient than even a bilateral extraperitoneal attack but the sympathectomy, especially on the right, is more difficult.

The incision is very much that of an approach to the kidney. Flothow recommends a muscle splitting one, dividing the skin almost horizontally just below the twelfth rib, then passing forward and downward, splitting the oblique muscles in the direction of their fibers (and transecting at least a few of the fascicles of the internal oblique). The transversalis and its fascia again are split horizontally, care being taken not to open the peritoneum. The operator then pushes retroperitoneally toward the vertebral column, lifting forward the lower pole of the kidney and the ureter (which he does not see).

The first landmark upon the surface of the psoas muscle, before the sympathetic is approached, is the genitofemoral nerve, a straight, tense, white filament. Beyond this, and close against the anterior part of the exposed vertebral bodies, lies the faintly pinkish ganglionated chain solidly attached by its fine rami.

On the right, the vena cava must actually be retracted to expose the chain. On both sides, the renal fascia is seen at the upper angle of the dissection. The renal artery can if necessary be felt.

Two rather spindle shaped but not strikingly marked ganglia will usually be found, the second and third lumbar, the latter just above the common iliac vein (on the right) or artery (on the left). Occasionally the two are fused. The ganglia and chain vary in size and their exposure may be made difficult by the extent to which the great muscles overlap them as they are applied to the vertebral bodies. The peritoneum must be held forward by gauze packing and a broad curved (lighted)

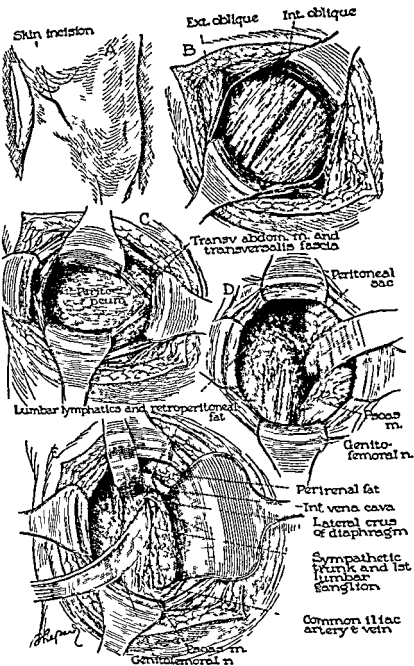


FIGURE 10 LUMBAR SYMPATHECTOMY—RETROPERITONEAL ROUTE In D, some tendinous fibers of the internal oblique (not shown) will have to be divided In E the ganglionated chain is too distant to show that the rami approach L 1 from above L, 2 horizontally and L 3 from below L 2 and 3 are often fused

retractor. It is best to sever the ramus first, then cut the sympathetic with scissors below the third ganglion. It can now be lifted up and divided above the second just below the renal vessels. To secure a sympathetic denervation which will carry the vasomotor paralysis as high as the thigh, the first lumbar ganglion must be included in the resection. In women, this is harmless; in men, it is very likely to upset the mechanism of

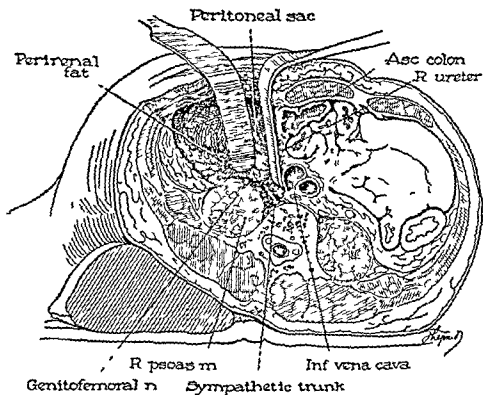


FIGURE 11 LUMBAR SYMPATHECTOMY. A cross section showing the retroperitoneal approach to the right sympathetic chain. The displacement of viscera, including the vena cava, is not exaggerated. The psoas muscle must be depressed to expose the sympathetic.

ejaculation. Removal of this ganglion in males should therefore be practiced with caution.

The wound is closed with silk in layers.

The result of sympathectomy should ideally be the establishment of warmth, dryness and freedom from vasospasm in the affected limb. Tests of the skin temperatures should show a degree of vasodilatation which can neither be increased by

any attempted reactive hyperemia nor appreciably diminished by moderate exposure to cold. In other words, the power of the peripheral circulation to display a vasomotor reaction in either direction should be lost. The condition should be permanent. In the legs, it usually is permanent. In the arms, under the operative system now in use, it will probably prove so to be. Strangely enough, persons who have lost their vasomotor control do not seem to miss it. They enjoy the sense of warmth in their fingers or toes and make no objection to the dry skin. Even if the procedure is not completely successful, patients are usually pleased with the result, and the individuals actually made worse by degeneration of the postganglionic vasoconstrictive nerves and exposure to spasm from adrenal influence (under the earlier method of treating the supply to the arm) are after all few.

**Pneumatic Hammer Disease**—This form of arteriospasm, though much like Raynaud's phenomenon, affects only male workers who use a rapidly vibrating stonecutter's hammer. Within a few months after first using the instrument, the workman may become subject to attacks of vasospasm in certain fingers. These attacks are excited by exposure to cold, particularly in the early morning or after the day's work is over—never while the hammer is actually being used. Between attacks the fingers show no change, nor is the disease progressive or destructive. Because of the manner in which the hammer is held, the fifth, fourth, and, occasionally, the third finger of the left hand are especially affected, sometimes the tips of those of the right hand as well. The disease was first identified (1917-18) by investigators for the United States Department of Labor and of Public Health. A good description is given by Hardgrove and Barker, from the Mayo Clinic.

#### SCLEPODERMA ACROSCLEPOSIS SCLERODACTYL

This disease, actually rarer than that of Raynaud, may possibly be a near relation. It is practically confined to young females, showing itself in childhood. Attention has been called

to the hardening of the subcutaneous tissue, shrinking of the skin and smoothing of the natural wrinkles in advanced Raynaud's disease. Such a state is known as Sclerodactyly. Scleroderma seems, in some instances, to be a more wide spread development of the local digital vasospasm and secondary sclerosis, in others, the general hardening of the subcutaneous tissues and atrophy of the skin appear to be primary. A form is actually described in which the skin of the body and limbs becomes hairless, thick and firm, the process spreading down the arms to the hands without causing vasomotor symptoms. In any case, the full fledged disease stiffens the fingers, forearms, and even upper arms, makes a mask of the face, smoothing out all natural folds and wrinkles, tightens and fixes the skin, particularly over the forehead and malar prominence, and finally, in many cases, leads to arthritic changes and ankylosis. The eyes and mouth are reduced in size. Neither can fully be opened, nor can the eyelids be tightly closed. The front of the chest is apt to be affected, the toes often, the feet only occasionally.

Microscopic examination reveals atrophy of the growing layer of the epidermis. The deep skin is fibrosed, the subcutaneous tissue as well. Indeed, fibrosis of soft tissue is sometimes so advanced as to extend into the muscles and bind skin to bone. The small arteries are embedded in scar, their caliber greatly reduced. This extensive hardening of the tissues may follow upon Raynaud like attacks of cyanosis of the fingers—in which case it follows rather promptly—or it may occur coincidentally with such attacks. It may even fully establish itself in the absence of any attacks whatever. It is idle to speculate whether arterial spasm is a prime factor or whether some basic infection or endocrine disturbance is at the bottom of the whole process.

The course of the disease, though often rather rapid in its early stages, during which the hands, arms and face are hardened, is decidedly chronic, dragging along for years toward a fatal ending. Once hide bound and stiffened by arthritis, the victim is in a pitiable and hopeless state. No one actually

recovers from the disease. The best that can be asked is that it should become stationary at a stage when the individual is not actually crippled.

*Treatment* is most unsatisfactory. When intermittent vaso-spasm is a feature, the patient can of course be protected from cold and other vasospastic influences, as in Raynaud's disease. The skin is softened and made as pliable as possible by massage and greasing. Nutrition is kept up as well as possible. Sympathectomy occasionally makes the fingers more supple and comfortable, though any decrease of stiffness is rather more likely to be due to diminished edema than to any increased pliability of the skin. It should not be used unless vasodilatation has been proved possible. If the metabolism is low, desiccated thyroid should be pushed.

#### ARTERIAL SPASM IN RESPONSE TO INJURY

Traumatic arterial spasm is brought on by a variety of injuries, many of them violent, such, for instance, as wounds of war and accident, bullet wounds, fractures, blows, stabs, and punctures. Sometimes there is evidence of direct trauma to the arterial wall, almost as often, surprisingly enough, the artery itself seems never to have been touched. Large arteries such as the femoral or the brachial have chiefly been affected, partly perhaps because of their length and exposed position. On the whole, it would seem as if the spasm were the result of a disturbance of the local vasomotor nerves, and at the beginning, at least, thrombosis is certainly not a factor. Spasm of this sort lasts for hours or even days and usually leads to no complicated after effects. There is another great group of arterial vasospasms, however, undoubtedly merging into this one, which is of a decidedly more chronic sort. In this, the spasm appears as a rule to be due to a vicious reflex, and the associated changes in the skin, nerves, muscles, bones and joints may overshadow the direct evidence of vasoconstriction. Included in this group of chronic arterial spasms are causalgia and the causalgia like states, reflex dystrophy of the extremities and traumatic osteoporosis. There are still other



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pin prick. Motions of the toes were feeble and painful. It was thought that all this was due to contusion by the bullet, causing an intense spasm of the femoral artery. Accordingly, the leg was massaged every fifteen minutes, and after nine hours the whole extremity became bright red and warm, the pulse returned and the normal sensibility was restored. The wound healed without incident.

Here, of course, are instances of contusion of an artery by a bullet which passed very close to it, in effect, a blow upon the vessel itself yet without any permanent injury or thrombosis. Other reports such as that of Kuttner and Baruch tell of examinations of such vessels and in one instance (a wound at the ankle) excision of a seemingly thrombosed stretch of posterior tibial artery. Whereupon the vessel, both proximal to and beyond the part resected, resumed its pulsation (retrograde circulation) and much to the operator's surprise, no thrombosis in the excised specimen was found. Thus additional information was secured, for it is evident that resection of the contracted vessel broke up the spasm of the vascular tree of which the posterior tibial was the main stem.

Instances of acute arterial spasm resulting from fractures are described by Montgomery and Ireland, from whose paper the following is abstracted.

A boy, four years old, had suffered a fall upon his left elbow an hour before coming under observation. There was found a fusiform, discolored swelling with lateral angulation of the arm below the site of injury and marked tenderness over both condyles of the humerus. Distal to the elbow, the skin was *dead white* and distinctly colder than that of the opposite hand and fore arm. No pulsation could be palpated in the left radial or ulnar arteries. The X ray showed a comminuted supracondylar fracture with lateral displacement of the lower fragments, the radius and ulna being dislocated posteriorly.

Four and one half hours after the injury, reduction under gas oxygen was carried out. The circulation remained unchanged.

states such as acute arteritis, seemingly related to an anatomic abnormality such as cervical rib, which are even less easily classifiable. All these are roughly sorted out below in the sections which follow. It is not necessary that all should agree as to their relation to each other.

**Acute Traumatic Arterial Spasm Arterial Stupor**—Though it would be impossible to tell of all the injuries which may excite traumatic spasm, several cases, due to bullet wounds, to fractures, to punctures and to blows have been selected as examples. It will be observed that the first cases cited are of a rather direct type of trauma to great vessels, and are marked by arterial stupor. Naturally, most observations of this sort have been made in war time. Two very instructive experiences are reported by Kroh (1915). The first is one of a wound of the right inguinal region by a revolver bullet. There had been a good deal of venous bleeding from the wound of exit. The femoral artery was pulseless. The soldier complained of poorly localized pain in the lower thigh. Exploration showed that the saphenous vein was severed. This was doubly ligated. The femoral arteriovenous sheath was seen to be infiltrated with blood. The femoral vein, fairly well filled, was identified but the artery could not be found for some time. Finally a pulsating thread, the size of a knitting needle, was isolated which should have been the femoral but seemed to be an anomaly! As it was watched, however, it gradually enlarged and much to the operator's surprise soon took on the size and pulsations of the femoral.

This observation evidently prepared Kroh for the following case which was treated without exploration. A soldier, who had been shot with a rifle bullet through the upper thigh, complained of numbness and tingling in his foot. The bullet entered near the anterior superior spine and came out just below the scrotum. The intervening skin was discolored with blood. The pulse in the femoral artery just below the track of the bullet was barely perceptible. In the popliteal and posterior tibial it was absent. The skin from the knee down was yellowish white, cold and damp, completely anesthetic, even to

Hence the multiplicity of names. Whether the basic exciting lesion is in the sympathetic or sensory nerves and just what reflex pathways are involved is still uncertain. Doubtless many sorts of injury, including blows, infected and uninfected wounds, even burns and frostbites, are able to set off the prolonged, serious, but usually reversible reflex disturbance. Although he invokes, even in one case, both a vasodilating as well as a vasoconstricting influence, Leriche has consistently laid the trouble to the vasomotor nerves, the peripheral sympathetic system. A typical case of his, of which another example is illustrated opposite page 148, is here quoted.

"Case 3. This man is a polish worker. After a severe blow on the left foot he presented inability and pain. There was no fracture. No treatment gave any relief. The patient stayed in bed, and for almost a year could not walk. He was finally considered to be a malingerer and sent to me with this diagnosis.

"Examination showed the whole left lower limb to be extremely painful. The slightest touch upon the skin made him cry out. No movement was possible. The leg and the foot were cyanotic. There was evident atrophy of the foot, the leg, the thigh, the buttock, and even the left side of the back. Radiography showed extensive decalcification of the foot and lower third of the leg.

"Considering this wide disturbance of his troubles, I performed a lumbar ramisection on Nov. 22, 1926. The same evening pain and cyanosis had gone. The following day feeble movements were possible. Sixteen days after operation, the patient got up for the first time for eight months. He could bear weight upon the foot without suffering. Functional conditions improved progressively. The bone recovered rapidly its calcareous matter, and successive roentgenograms showed a very regular reconstruction of the bone which was absolutely normal after three months. At the end of March, 1927, the patient was discharged perfectly cured."

In other instances of this general type, Leriche makes a great point of the traumatic arthritis and hydrops of joints

Incision was then made into the antecubital region where a large hematoma was found and evacuated. The brachial artery was seen to pulsate down to a point an inch proximal to its bifurcation, but here all pulsations abruptly ceased "*as though the blood were striking against a solid structure*". The pulseless arteries were contracted, did not appear to have been injured in any way and seemed not to be thrombosed. Warm, wet dressings were applied to the open wound.

On the day following, the hand felt warm and the nails were no longer completely blanched, but the pulses were still absent. Three days after operation, a faint radial pulse returned, *slowly improving in quality until it became altogether equal to that of the uninjured arm*. Eleven days after operation, the wound was closed and a posterior cast applied. However, no attempt at final reduction was made at this time. Indeed it was not until fifty three days after the injury that complete replacement by an open operation was made. This caused no circulatory upset.

It is not easy to say whether this spasm (stupor) was induced by direct trauma to the artery, of which the hematoma perhaps offers some evidence, or whether injury to its sympathetic nerve supply was the cause. Perhaps it makes very little difference, since, in any case, a local nervous mechanism in near relation to the vessel would seem to have been involved. However, *such an arteriospasm is a direct result of the bullet wound, fracture, or whatever, and not a reflex disorder, as seems to be the case with the more obscure and complicated group which follows*.

**Reflex Arterial Spasm** **Chronic Segmental Arterial Spasm** **Causalgia** **Reflex Dystrophy of the Extremities** **Trophic Edema** **Traumatic Osteoporosis**—Although this group is a loose one and although observers, dating back to S. Weir Mitchell, may perhaps have been describing a considerable variety of states, there has been for some years a fair unanimity of opinion that there is such an entity as prolonged reflex arterial spasm and that this is capable of causing disorders of skin, connective tissues, nerves, muscles, joints and bones.

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resulting from what he believes to be reflex *vasodilatation*. Such leave behind many stiff joints. Whether or not he confuses the passive congestion of vasoconstriction with vasodilatation is immaterial. The disorder of joints, bone and muscle (atrophy) presumably go together as a rare response to even rather mild injuries.

Another somewhat similar type of disease but now due to an infected wound is described by Lehman. D E D, a school girl of eighteen, had suffered, four months before coming under observation, a punctured wound by a crabapple thorn upon the ulnar border of the right forearm. This had become infected and had been subjected to exploration and drainage. A sinus had been left surrounded by induration, heat, and redness half way to both wrist and elbow. The patient feared to have the forearm touched and could barely move the wrist and fingers. During the following seven months, four operations for drainage were performed. Almost a year after the original injury, the forearm and upper arm were swollen and doughy up to the axilla. Pain was continuous. The fingers were cool. The right radial and brachial arteries pulsated (to the touch) less strongly than those of the left hand. Though one of the old incisions was unhealed, the temperature and leucocyte count were normal. Amputation had been suggested.

On the basis that the condition resembled the "trophic edema" of some war wounds and because tissue removed at the last operation had shown a perivascular inflammation, a periarterial sympathectomy was performed upon the right brachial artery. Two days after the operation, movements of the fingers had improved and tenderness of the forearm had diminished. Six weeks later, motion at the elbow was free and that of the fingers was beginning to improve. Swelling had disappeared and the skin had become warm and dry. The wound had healed. In six months, except for some weakness, the extremity was normal.

This case appears to have been one of a rather diffuse vaso-spasm in response to a local injury and infection which probably did not actually touch any large artery or nerve. The

vasospasm mounted well above the lesion and affected both motor and sensory functions. The reflex disorder, for such it must be called, was broken up by removing the nerve fibers surrounding the brachial artery. It would seem that a cure was due to the interruption of stimuli going toward the central nervous system, since the interruption of sympathetic impulses passing out upon the brachial artery could hardly have affected parts of the arm proximal to the operative field. This case, in respect to its painful state and oversensitive skin, much resembles causalgia, an instance of which is taken from the original publication of Mitchell, Morehouse, and Keen (page 107).

"Case 24.—Hiram Weston, æt 42, Co E, 18th Mass, enlisted May, 1861. Healthy until wounded, in the Wilderness, Mar 5, 1864. He was running at a double quick, and was shot in the left arm. The ball entered three and a half inches immediately above the internal condyle of the humerus. It emerged directly below the anterior angle of the axilla, two and a half inches lower. The ball passed over the nerves, and injured the ulna nerve especially.

"He felt violent pain throughout the limb, which was instantly flexed at every joint and so continued for fifteen minutes, when it was extended by the aid of the other hand. The pain which then began never left him. The arm soon lost motion entirely but within a few days regained so much as it has now. As to sensation, he can tell us nothing, except that probably it was only damaged in the ulnar range of nerve supply.

"Present state.—It is now fifty days since this man was shot. Of the intervening period, he gives a very clear account. Immediately after the wounding, the whole limb swelled, but this rapidly subsided and the hand was no larger than its fellow until about the fortieth day, when it became rapidly oedematous. The pain has consisted all along of darting pangs from below or under the elbow, down into the hand, but not on the anterior surface of the forearm. In the hand, the pain is burning and tingling, or as he calls it 'prinkling' . . . The



hand is sore to touch everywhere, but tact is unimpaired, save in the little finger and ulnar side of the ring finger \* \* \* The nutritive changes did not become marked until about the forty fifth day, they are now obvious, and in time will doubtless give rise to the glossy skin, to which we have so often referred. The hand is swollen. The palm is red and dotted with patches of thickened epithelium. \* \* \* The nails are laterally much arched, the skin at their bases retracted, \* \* \* The joints are exquisitely tender, and very stiff and swollen. The patient has kept the hand wet ever since he was hurt \* \* \* Tactile sensation is perfect throughout the hand, except in the fourth finger and the ulnar side of the third."

The authors feel the case to be typical of the symptom complex which Mitchell later named *causalgia*. The burning pain comes first, then the glossy skin. But the pain itself only arises during the healing of the wound. Moreover it may transfer from the field of the wounded nerve to that of an unwounded one. Its site is always the foot or hand and here the nutritive changes are seen. The part affected is not only subject to the intense burning but is so sensitive to a touch or draft of air that the victims almost invariably keep it moist.

The foregoing description, which does little justice to the full account of Mitchell and his associates, fits fairly well the chronic arterial spasm and reflex dystrophy of today. Into the story of Hiram Weston one may read, if one likes, an ulnar nerve injury, a blow upon the arteriovenous bundle followed first by a venous thrombosis and, after some forty days, by the full fledged edema, malnutrition, and oversensitiveness of a serious, extensive, reflex arterial spasm. One can find in the writings of Meige and Madame Athanassio Benisty descriptions of *causalgias* resulting from wounds received in the war of 1914-18, exactly similar to those of Mitchell. But the French neurologists call attention to the fact that Weir Mitchell described particularly wounds of the brachial plexus and failed to notice the *causalgias* of median and sciatic nerve injuries. They themselves describe these states very accurately. They say

"In the painful form of wounds of the median nerve with major cruralgia, the hand takes on an emaciated aspect, the skin is delicate, wrinkled, rosy in color, and marbled with bright red patches, it is hot both subjectively and objectively. The arterial pressure is elevated. Sweating is abundant. Nutrition is decidedly changed."

They find that the nerve itself, when exposed, is congested and that the vasomotor and sudomotor disorder is shown principally in the structures supplied by nervous terminals of the sympathetic, that is, in the various nervous (Pacinian) corpuscles and the capillaries of the skin. They regard the median and sciatic as especially vulnerable because they are rich in sympathetic fibers and are well supplied with vessels which themselves are abundantly furnished with vasomotor nerves. There is probably a distinction, which has never been clinically clear, between the cruralgia of median or sciatic origin, that is, a primarily nervous lesion on the one hand, and on the other, the sort of injury which Leriche was the first to cure by interruption of the periarterial nervous pathways. Both his early cases were wounds in the region of the upper axillary artery. In one, he stripped the axillary and later resected, with a very favorable result, its proximal portion. His second case is briefly described below.

Corporal G. was wounded Sept. twenty fifth, 1915, by a bullet which fractured his left clavicle. A flaccid paralysis of the left arm made it seem probable that the brachial plexus had been divided. Ten days later, Leriche explored the plexus, finding it and the distal part of the subclavian artery embedded in scar tissue. The plexus had not actually been injured. There were no pulsations below the clavicle. He dissected free the plexus and vessels without effect. The hand remained cold, deeply cyanotic, and totally paralyzed.

Five months later, the soldier reported back to Leriche. The hand was colder and more purple than ever. The skin was glossy and felt cold like that of a snake (by contrast with some of the median and sciatic nerve injuries in which the extremity is hot). There were blood blisters on the fourth

and fifth finger-tips. Crises of burning pain kept recurring in the hand. Leriche now laid bare the upper brachial artery, which was only two to three mm in thickness, did not pulsate, and was covered with reddish patches. The neighboring nerves were soft. Stripping off the outer coat of the artery caused no bleeding whatever. But on the following day the soldier noticed tingling in his whole arm and felt much relieved. The left hand, previously colder than the right, had now become the warmer of the two. The blisters dried up. Three weeks later some motion was beginning to return in the forearm. No further account is given.

Leriche believes that the wide-spread arterial spasm broken up by periarterial sympathectomy accounts for the paralysis and atrophy in such cases, no actual lesion of the great nerves being present. Yet the vasomotor disorder seemingly is capable of affecting the peripheral nerves through their blood supply, causing paralyses and weird contractures. The fingers, for instance, may be left extended but pointing together, the thumb flat in the palm, the wrist straight or flexed. Such a deformity somewhat resembles that of paralysis of the median nerve above the elbow. With it, the trophic disorders so often seen—blisters, ulcerations, desquamation, deformed nails—are consistent. Probably some causalgias or causalgia-like states are primarily due to peculiar peripheral nerve injuries, while others are the result of irritations of the plexus of nerves surrounding the great arteries of the limbs. In the latter case, vasomotor spasm, if sufficiently prolonged, may cause a great variety of nutritive changes in bones, joints, muscles, subcutaneous tissues and skin, imitating in some cases, if it does not actually occasion, serious inflammation of certain great nerves. Apparently a vicious sensory sympathetic reflex is set up, as pictured by De Takats. Indeed, some such mechanism must be imagined to explain the dramatic cures so often secured by blocking the periarterial nervous pathway or excising the sympathetic ramus and ganglia.

Minor degrees of these reflex disturbances are rather common. It is not necessary that all or indeed many of the peculiar

changes described should be present. A little edema, a little alteration of superficial sensibility so that scratching or handling the part is unpleasant, a diminution of the peripheral pulse, a moderate atrophy of the bones, such will often be noticed after a variety of disorders ranging from serious fractures to cat bites and from laceration of a great nerve to a superficial bruise. The condition may show a tendency to spontaneous recovery, but as a rule it is decidedly persistent. The best test of its presence is a paravertebral nerve block (or spinal anesthesia). This will usually, for the moment, bring on a full vasodilatation, restore the natural sensibility of the skin, and even diminish edema. It will guarantee a favorable result from sympathectomy, provided the seriousness of the symptoms demands such a step.

**Treatment**—In all reflex osteoporoses, reflex edemas and causalgia like states, the effect of paravertebral nerve block with procaine should be studied. In the milder cases, hypersensitiveness of the skin will not only disappear for as long as the block lasts, but will sometimes, from that moment, show a progressive improvement. It may therefore be advisable to repeat it. Relief is favored by the skillful use of massage, heat and even psychotherapy, in fact, by any means of relaxing vasoconstriction. It is conceivable that drainage of an infected wound may be curative. But in the more serious cases, a sympathetic neurectomy is required. Obviously a paravertebral ramisection or ganglionectomy will break the outflow of vasoconstricting impulses, and there are very few cases incurable by this means. However, as already explained, even a "periarterial sympathectomy", that is, stripping off the outer coats of the principal artery supplying the affected limb, which presumably acts by interrupting mainly central going impulses, will often break up the vicious reflex. This was Leriche's original contention and is well demonstrated by Lehman's case. The whole matter will come up again in the management of the remarkable arterial spasms which are so often associated with arterial embolism and even with venous thrombosis.

**Acute Arteritis Cervical Rib Scalenus Anticus Syndrome**

(Naffziger Ochsner).—There is some question whether all these terms refer to the same abnormal sort of arterial spasm. Acute arteritis may perhaps be a variety of arterial stupor or even reflex chronic arterial spasm. It will be sufficient to present it as occurring in the brachial artery. Although infection at a distance has been invoked as a cause, it is held here that some unnatural relation of the brachial plexus and subclavian artery to the first rib or an actual cervical rib is more likely to be responsible.

*The Clinical Signs* of abnormal pressure upon or irritation of the brachial plexus and subclavian artery usually point to a nervous rather than to an arterial difficulty. Indeed, Naffziger regards the arterial disorder as relatively rare. The syndrome is more common in women than men.

The principal nervous symptom is pain, referred to the suprascapular region and shoulder, the side of the neck and ear, the arm and forearm, especially upon the ulnar surface. It may be tingling and numb or sharp and knife like. It is usually aggravated by depression of the shoulder. Suprascapular tenderness and a radiation of the pain down the ulnar side of the arm are often brought on by pressure over the scalenus anticus muscle at the root of the neck.

The first vascular manifestation is usually weakness of the arm, made worse by exercise. Then, coldness, numbness and tingling gradually set in. An early diagnosis is difficult. Relief of the distress by elevating the shoulder and aggravation of the symptoms by lowering it are confirmatory. The radial pulse may be obliterated or its weakness may be evident, or oscillometry alone will show that pulsation is slightly lessened in the affected arm. The brachial artery is occasionally felt as a tender cord, proximal to which the axillary and subclavian beat normally. If necessary, arteriography can be used.

In a case described by Clute the following observations were made. The patient, E. K., a man, thirty five years of age, had suffered for several years from a nonspecific prostatitis. Six months before coming under observation, he had noticed blanching, coldness, and occasionally cyanosis in the left hand

and arm Pain was moderately severe and had progressed upward along the radial and brachial arteries The left arm was found to be cold, damp, and cyanotic On motion, the fingers blanched The brachial artery could be palpated as a deep, tender, swollen cord There was a good pulse in the axillary, none below

Clute first explored the supraclavicular region The subclavian artery and brachial plexus did not appear to be pinched between the scalenus anticus muscle and the first rib Nevertheless, a few of the fibers of the tendon were divided No improvement followed.

The brachial artery was then explored and found to be a firm cord surrounded by a mild edematous reaction Two inches of the artery and vein were resected The vein proved to be normal, the artery, the seat of "chronic periarteritis" There was no thrombosis and no bacteria could be cultivated from the tissues Improvement set in within three days and ended in almost full recovery of all functions The arm was only slightly disabled but the radial pulse did not return

Here is a disease which is not associated with injury, with which infection can have had little to do, and which may fall into the class described by Telford and Stopford (who attribute the explanation of the condition to Todd) namely, that either a first or a cervical rib is capable of making pressure upon and irritating sympathetic fibers entering the lower cord of the brachial plexus for distribution to the brachial artery (rather than the subclavian or axillary) The initial vasoconstriction causes pallor and coldness Later, they say, the vasa vasorum are obliterated and finally the brachial becomes thrombosed. They feel that it is not so much the abnormal position of the rib as a peculiar exposure of certain sympathetic fibers to injury which causes the trouble The periarterial filaments are in the state of irritation which Leriche has so vividly described by saying that the artery has become "a diseased sympathetic nerve"

It is perhaps unfair to suggest that the particular case just described is really one of an unnatural relation between the

upper rib and the brachial plexus. Yet the signs and symptoms are those characteristic of the circulatory disorder caused by a cervical rib or the scalenus syndrome of Naffziger. The truth is that only after many years and because of repeated lowering of the shoulder or bending of the head, does the necessary nervous irritation due to contact between rib and the lowest part of the plexus take place. A cervical rib may be present on one side and the symptoms on the other! Ochsner finds that in many cases the scalenus anticus appears to have become unnaturally shortened (by repeated irritation of the nerve supplying it) and to have lifted the cervical or first rib unnaturally high. Thus a vicious circle is established which can be broken by dividing the tendon of the scalenus anticus and letting the rib drop. Adson and Coffey had already suggested this step but with the idea of letting the artery and plexus slide forward and downward. This matter will again come up in a consideration of treatment. When exposed, at operation, the subclavian artery, *distal to the rib*, has often been noted to be dilated, almost like an aneurysm. The dilatation is presumed to be related to the constriction of the brachial beyond, which, by the time the signs of the disease are advanced, may have become impermeable because of contraction or actually thrombosed.

*Treatment* —When the signs persist, even though the use of the arm in a hanging posture is prevented and in spite of sleeping with the arm suspended above the head, operation is indicated. The supraclavicular region is approached as in the anterior operation for cervico thoracic ramisection (page 127) and the subclavian artery can readily be inspected, but it is not easy to be sure that the supposed nervous irritation is taking place unless a cervical rib is present, the first rib is held very high, or the subclavian artery is enlarged. In any case, the phrenic nerve can be drawn aside and the tendon of the scalenus anticus divided. A lowering of the rib may be the only obvious change secured, in which event the wound may properly be closed without further surgery, to determine the effect of the procedure. In an early case, the brachial artery

will, at once or in a few days, relax. If it remains a contracted cord, it had better, as Leriche directs, be resected for several inches. Whether or not the radial pulse returns, the symptoms are then likely to be relieved.

### PERMANENT STATES OF VASOSPASM

Up to this point, the arteriospasm described have been in intermittent, temporary or prolonged, yet as a rule not strictly permanent. Those now to be considered are of a permanent sort. Some are due to such disabling diseases of the central nervous system as infantile paralysis. Others are very similar permanent states of vasospasm for which no cause can be named. Why some of these vasoconstrictions should result in ulcerations, some in trophic disorders, and others merely in blue, sweating feet and hands, by contrast with the vicious causalgias just described, is as yet not understandable. One can only keep in mind the simplest form which arterial spasm may take, and recognize the variations upon it.

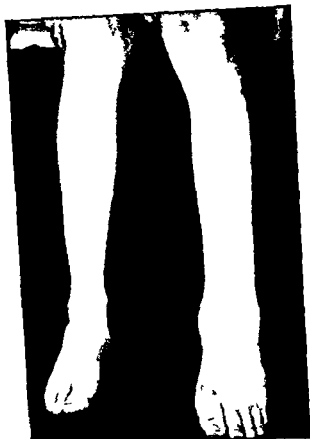
**Infantile Paralysis Spastic Paralysis**—Children born with spastic paralysis (Little's disease) or who have recovered from anterior poliomyelitis are often left with atrophied, purplish, mottled, cold, damp limbs. That this unpleasant state is due to arterial vasospasm is proved by the vasodilatation which results from sufficiently warming the body and by the aggravation of the discomfort which follows exposure to cold. In other words, the trouble is permanent, but is reversible by appropriate means. Whether the vasoconstriction is due primarily to disuse or to a neurogenic disorder is not clear. In any case, it is curable by a properly performed sympathectomy. This was first brought out by Royle's operation, originally a ramisection, intended to diminish the tonicity of the muscles in spastic paralysis. The treatment failed in its principal object but was discovered quite unexpectedly to have left the paralyzed limb warm, pink and dry. Actually it opened the way for the general use of sympathetic ganglionectomy to relieve arterial spasm. Not only is the blue, cold extremity made pink and comfortable but if the atrophy of bone and soft parts has not



existed for too many years, the limb, whose length and girth have lagged far behind its mate, may show an acceleration of growth. The procedure is also of use in connection with plastic operations on the foot, encouraging the healing of wounds. To determine that the operation is indicated it is only necessary to use one of the tests of vasoconstriction described in Chapter I.

**Acrocyanosis**—Like Raynaud's phenomenon, this is rather a physical sign or symptom complex than a disease. The name merely means blueness of an aeral part, almost necessarily an extremity. The arteriospasm which causes the blueness is permanent. The hands and feet alike—in this case the feet rather more than the hands—are continuously blue, cold, and sweaty. Probably in different parts of the world the condition varies in this or that detail. Crocq, who named it (1896) described it as occurring in young hysterical women. It is certainly rather common in girls, especially Jewish girls, appearing at about the time of puberty. Some of these individuals suffer from pes cavus as if there were some slight congenital background such as a spina bifida occulta. But the same state or one indistinguishable from it may crop out in persons of any age, men as well as women. There is one particularly troublesome form which has been called *Erythrocyanosis Frigida*. Telford and Simmons, in their excellent account, say that this is known on the continent as erythrocyanosis crurum puellarum and in France as erythrocyanosis sus malleolaire (above the ankle). Its remarkable feature, as will presently be told, is ulceration of the lower leg which takes a most disabling and intractable course.

Acrocyanosis in its mildest form is very familiar, in its serious form, rare. Once established, it seldom altogether disappears, though the adolescent sort tends to be outgrown with maturity and in many individuals is only really troublesome in cold weather. The degree of redness or blueness varies with the vascularity of the particular skin. The change is by no means confined to the digits, for the whole hand is discolored, the whole foot and even the leg for some inches above the



TRAUMATIC EDEMA (REFLEX DYSTROPHY) Following an injury to the left foot without fracture. The atrophy of the bones of the foot (roentgenogram) was extraordinary. The skin of the foot and leg displayed hypesthesia and advanced hypersensitivity to scratch or pinch. Six hours after a diagnostic spinal anesthesia the edema had nearly disappeared. (Courtesy of Dr. John B. Cross, Atlanta.)



**ERYTHROCYANOSIS IRRIDIA, A SPECIAL FORM OF ACROCYANOSIS.** On the left the preoperative state, the ulcers are open the skin of the feet and lower legs dark (reddish blue). On the right, the postoperative state the ulcers healed, the skin of natural color.

ankle As one passes a hand down the leg from the knee, a coolness is usually encountered perhaps half way to the ankle, a coolness which, in many cases, becomes actual coldness as the ankle is reached There is very little discomfort, but the cold feeling may be very disagreeable In damp cold climates, chilblains occur

*Erythrocyanosis Frigida*—The acrocyanosis of young girls may take this serious form The trouble shows itself chiefly in the feet and legs, overshadowing the moderate blue dampness of the hands It is always bilateral Not only the feet but the lower half of the legs are purplish, especially on the posterior surface In a fair proportion of cases, ulcerations occur Anywhere between the malleoli and the mid-calf, indurated nodules appear and slowly break down into ulcers There may be two to a dozen such sores which are of a moderate depth and hardly exceed one cm. in diameter The lesions are distinctly reminiscent of erythema induratum, or Bazin's disease, but while the latter occurs upon the front of the thigh, cold legs of ill nourished girls and is believed by many to be a form of tuberculosis, erythrocyanosis frigida attacks well nourished, even fat girls The ulcers of both, however, are equally chronic and difficult to cure The diagnosis is not at all difficult and the cyanosis and coldness are readily driven away for the moment by inducing a reactive hyperemia. The pulses are quite normal, the vascular spasm taking place presumably in the arterioles This is in contrast with the recurring spasms of Raynaud's phenomenon in which the digital arteries are the scene of the vasoconstriction Plate V, opposite, pictures the case of M MacL., described on page 151

*Treatment* of acrocyanosis need seldom be radical unless the sense of coldness is very troublesome or ulceration occurs If any deformity of the foot exists, a spina bifida occulta should be looked for but can rarely be treated directly with success Apparently, like Raynaud's phenomenon, acrocyanosis is aggravated by psychic upsets as well as cold, so that protection in these directions is required Though the milder cases, if they occur in young girls, will usually become less trouble-

some with advancing years, the serious and especially the ulcerated states should be subjected to lumbar sympathectomy. The results of this operation are excellent.

The following are instances, respectively, of a mild and of a serious form of the disease.

B Y, a Jewish girl, thirteen years of age, complained of cold, blue fingers and toes. The catamenia had been established a year earlier. The child was of unusual intelligence, played the violin, ate her meals in a hurry, and got insufficient sleep. Her nutrition was good.

For three years, the coldness, dampness, and cyanosis had been increasing in both feet and hands. The feet had given the most trouble, apparently because the little toes had progressively overlapped the fourth toes, finally projecting so far dorsally that another surgeon, two months earlier, had amputated both. The balls of both feet had become prominent, the arches high, the proximal phalanges extended and the distal joints flexed. The child was aware that any emotional strain—her violin lessons, seeing a doctor—made the coldness and sweating worse.

The hands were reddish blue in color, cold and damp. The change toward normal came rather gradually at the wrist. The toes and feet were more blue than red. The legs were dusky. Coldness was marked up to a rather definite level just above the ankle. No changes in the subcutaneous tissues were present and no ulcerations. Studies of cutaneous temperature were not made.

The patient was advised to eat slowly, rest after her midday meal, secure at least nine hours of sleep and protect her hands and feet from cold. When seen two years later, the feet were less troublesome, the hands were about the same. Sweating was less noticeable.

Seven years later, at the age of twenty, the patient made no more complaint of her feet. Both fingers and hands were reddish blue. Their palmar surface was glistening wet (when she came in for examination) yet the rest of the body sweat no more than seemed natural. The patient, without feeling that

her life was miserable, would have welcomed any operative relief

M MacL., a girl, seventeen years of age, complained of recurrent ulcers of both legs. For the last few years, these had broken out with the first snow storm and had disappeared in the spring. Her feet perspired more readily, she thought, than those of others and frequently felt subjectively cold. She appeared for treatment in March.

She was a good sized, somewhat obese girl. Examination of all systems and organs was negative save for the legs. The hands were perhaps a little red, but not beyond familiar limits. The blood pressure was 95/60. Both legs, for their lower two thirds, were mottled and reddish in color. Just above the ankles were half a dozen ulcers on each leg. These were much alike, shallow, round, only a little moist and surrounded by a red tender areola about one to two cm. in width. The feet and toes resembled the legs but were not ulcerated. Both dorsalis pedis arteries pulsated normally.

At a room temperature of 27° C (80° F) the temperature of the great toes was exactly that of the air and somewhat lower than that of the skin at the upper margin of the ulcerated area. Here the temperature was about 29° C (85° F). Spinal anesthesia brought about a maximal rise upon the great toes, to 34° C (95° F) and upon the mid leg to 32° C (90° F). This reaction was considered favorable for relief by bilateral lumbar sympathectomy and the operation was performed transperitoneally. The immediate result was entirely satisfactory. The ulcers promptly healed and the temperature of the skin remained high, being little affected by local or general exposure to various environmental conditions. The late result will not be known until several winters have passed.

#### HYPERIDROSIS

In describing the various states of vasomotor spasm, damp or even wet hands and feet have usually been described as associated disorders. Sudomotor activity is in fact almost in-

variably associated with sympathetic vasospasm, especially the persistent sort aggravated by cold and emotional stimuli. In some cases, the vasospastic blueness and coldness is more and the sweating less, but with the sort of hyperhidrosis which is really disabling, the cyanosis is less and the sweating more. When the hands and feet sweat excessively they very rarely flush red at the same time, there is almost inevitably some vasoconstriction.

Clammy, or wet, hands and feet are more than a nuisance. They are actually disabling. The slightest nervousness makes the skin glisten and some water actually flows. White, in an excellent account, tells of some special cases, of his own and others, of a man who felt he could not practice law because he must so often shake hands, of a medical student whose rubber gloves filled with sweat at the operating table. The change is limited rather sharply at the wrist and ankle, and the individual perspires no more over the rest of the body than do others. Both sexes are equally affected.

*Treatment* by any other means than sympathectomy is very unsatisfactory. Formalin (five per cent) soaks, macerates and irritates the skin. Radiation, if just the right exposure is used, causes atrophy of the sweat glands at the risk of a chronic dermatitis. Sympathectomy is especially suitable because after the sympathetic pathways are blocked, the glands can not be excited to secrete by adrenal influences (apparently they respond only to the chemical action of acetylcholine). Thus, even if the postganglionic neurones should be destroyed, the sweat glands will not be exposed, as are the arterioles, to the action of sympathomimetic hormones.

The appropriate operations to denervate the sweat glands of the hands and feet are exactly those advised for vasospastic states and described earlier in this chapter. In the thoracic sympathectomy, the stellate ganglion should not be disturbed lest Horner's syndrome set in, and in the lumbar operation it is sufficient to remove the second and third lumbar ganglia and the connecting chain. Unless the operator is very expert, the lumbar operations had better be carried out at separate

sittings The same is even more true for the thoracic sympathectomy The results should be permanent

### ARTERIAL EMBOLISM

When an embolus plugs an artery it is almost invariably true that it has come from a diseased heart Thus the background of the most sudden closure to which a great artery can be subjected is unfavorable to life As a rule, the heart is fibrillating, the left auricle dilated as in mitral stenosis, but sometimes, because of a coronary infarct, part of a thrombus is detached from the left ventricle Actually, any dilated heart, in the presence of congestive heart failure, may be a source of arterial embolism Very rarely indeed, the cardiac disease is so little marked that the source of an embolus can not be surmised

Emboli tend to lodge where a great artery divides The bifurcation of the common femoral at the groin is the most common site, nearly forty per cent of all lodgments occurring here The bifurcation of the common iliac and the aorta between them add about twenty five per cent more Thus sixty to seventy per cent of all emboli are more or less accessible from the region of the groin The popliteal division adds another ten per cent The rest occur in the arm, principally in the axillary or the bifurcation of the brachial.

If an embolus merely lodged at one of these likely spots, obstructing the vessels here and nowhere else, the result would be bad enough (it has been estimated that perhaps one in five limbs would be lost from a pure occlusion in the common femoral and twice as many from one in the popliteal) but closure of the artery by the embolus is not the whole story the arterial tree beyond the plug is thrown into spasm and thrombosis is often propagated from the point of obstruction far down the vessel

A classic description of the lodgment of an embolus tells of a sudden agonizing pain in the limb, coming without warning, and not due to the impaction of the embolus itself but to the resulting ischemia Such an event, coming out of a totally clear



sky, is not, however, invariable. Often there are prodromal signs, due either to small warning emboli or to the incomplete obstruction of the artery by the final large one as it is molded in the vessel. These signs take the form of a feeling of numbness or of tingling and coldness, the sort of thing one might expect with the onset of thrombosis, and the exact moment at which an embolus becomes lodged is not always easy to fix. Pain, however, is the rule and is usually severe enough to indicate the hour from which time elapsed after the complete obstruction can be counted. There is a great difference between embolism in the leg and in the arm. In the latter case, the initial coldness, or it may be pain, is severe, and though the forearm and hand, in embolism, become cadaveric, gangrene of any consequence seldom follows. In the former case, the situation is different. In middle age and beyond, the arteries of the leg are never as elastic, nor is a collateral circulation so easily established, as in the arm. Thus the lodgment of the embolus is followed at once by coldness and a cadaveric appearance of the foot and more or less of the lower leg. When the stoppage is such as to have allowed some blood to push, for a time, into the periphery, there is apt to be edema and a good deal of blueness. A considerable amount of blood may remain in the small veins. This will give a false impression of vascularity, for a pressure spot will quickly become colored again and even a small vein will refill (from the peripheral direction) after being emptied by pressure. Seldom is the foot of arterial embolism altogether white. It is always more or less evanotic and if the color be compared with that of the opposite leg, it will usually be clear that the change mounts well above the ankle, in rare cases into the thigh. Within an hour or two, the upper level of coldness will also be evident. If this is at or above the middle point of the lower leg, the prognosis is poor for survival of the foot. If it is merely just above the ankle, the level which marks the upper limit of vasoconstriction in the foot, both foot and leg may well be viable.

Bad signs in arterial embolism are, then, coldness, cyanosis

and edema, especially if these mount well toward the knee and if all pulses below that in the common femoral are absent. Good signs are some preservation of pinkness (however faint) and warmth, preferably in the foot, but at least as low as the ankle. The level at which pulsations are felt demands special consideration.

Lodgment of an embolus at the femoral bifurcation obliterates all pulses below the groin, that is, the femoral in Scarpa's triangle and below, the popliteal, posterior tibial, and dorsalis pedis. Just below the inguinal ligament, a vigorous pulse will be felt in the common femoral. The artery may even be palpably thickened. Both pulse and thickening may be due to the embolus which transmits a strong arterial beat from above. Lodgment of an embolus at the common iliac bifurcation may or may not completely obliterate the femoral pulsation. However, it always weakens it and sooner or later obliterates it altogether. It is not unheard of for an embolus to catch and then slip down, or, after lodging, to set up a thrombosis, finally plugging the artery after only partly closing it at first.

Lodgment at the aortic bifurcation generally blocks one arterial tree more than the other, but there will usually be evidence of some diminution of pulse in both femorals.

In determining the extent to which an arterial pulse extends down the leg, an oscillometer is valuable, but even without it a blood pressure cuff is helpful. If any oscillation can be detected at a particular level, it may of course be due purely to a collateral circulation. However, it is not particularly important, so far as the life of the leg and foot is concerned, to distinguish between a pulse transmitted through the main artery and one derived from collateral vessels. The main thing is to know whether or not a good pulsation is present in the mid calf.

To bring out some of these points, the condensed histories of several cases of embolism to the lower and upper extremities are quoted from the records of the Peter Bent Brigham Hospital.

M E F, Surgical 27614, a woman, thirty four years of age,

suffering from rheumatic heart disease, in the form of mitral stenosis and aortic insufficiency, had been reasonably well until the onset of the illness which brought her under observation. Three days before this moment, she had suddenly been struck down by a violent, almost unbearable pain in the pelvis which radiated down the inside of both legs, especially the left. Her vision was blurred, she vomited, and became semi-conscious. Both feet turned bluish white. Her left leg and foot were cold, numb and pulseless. Three days later there was still only a feeble pulse in the common femoral. Yet nothing worse than a purplish area outside the left heel had appeared. In the right leg, all pulses had returned. In two more days, an effective collateral circulation must have become established, because pulsation reappeared in the left foot and the patient recovered. A rider thrombus evidently caught on the aortic bifurcation, only to slide down into the left femoral. The patient's youth and dilatable vessels saved her leg.

F J McK, Surgical 59780, a spare man of twenty seven, suffering from rheumatic valvular disease, had been fibrillating for several days when he was seized with an agonizing pain in both legs. Within fifteen minutes he was seen by the surgeon. Pain had then settled in the right leg, which was white, cold and pulseless up to the level of the common femoral at the groin. Here a distinct pulsating thickening could be felt. Feeble pulses were present in the left foot. The patient was groaning in agony. Within two hours of the accident, the right femoral bifurcation had been fully exposed and an embolus was found in the common femoral projecting into the superficial branch. While all approaches were controlled by soft rubber tubing, the femoral was opened, the embolus milked out from below and extruded with a gush of blood when the current was let in from above. Repair was followed by complete restoration of the circulation. The moment the current was allowed to flow back into the peripheral vessels—and not before—the pain ceased. The operative procedure used is shown in Figure 12, page 160.

F V D, Surgical 38637, a man of forty seven, suffering

from a serious mitral lesion, entered the hospital fibrillating. Two days earlier, infarction of the left kidney was thought to have occurred. At three o'clock in the afternoon, he complained of severe pain behind his left knee and of numbness in the leg. That evening, pain and numbness left him but in the early morning, twelve hours after the initial attack, his pain recurred more violently than before and by noon had become excruciating. This was nine hours after the second episode. The left leg was then cold, bluish white in color, and pulseless up to a point just below the inguinal ligament. In this case again an embolus was removed from the femoral bifurcation, but at this late hour was adherent. A long, soft clot was found attached to its distal end. After removal and repair, a feeble peripheral pulse persisted for only a few hours. Evidently thrombosis occurred and gangrene followed. Embolectomy was performed too late, partly because the early symptoms were misleading.

W G B, Surgical 37369, a man, sixty four years of age and suffering from rheumatic heart disease, entered the hospital fibrillating. Four and a half hours earlier, numbness, which rapidly changed to pain, had attacked his left hand. The arm was held flexed at the elbow, the fingers clawed, cold and white, the arm cyanotic, distal to a point just above the elbow. When the arm was placed at a right angle with the body, a pulse could be felt in the axillary just distal to the edge of the pectoralis major muscle but not beyond.

Exploration, about five hours after lodgment, disclosed a solid whitish embolus, three cm in length, fixed in the upper brachial artery. To its lower end a filmy red clot, two to three cm in length, was attached. The artery was washed out with a citrate solution and seemed to be clear. Yet after repair, though a pulse could be felt in the lower brachial, the radial at the wrist did not pulsate. However, the forearm and hand were reasonably pink and the patient made a good recovery.

A year later, he was found to be doing well. The radial pulse had returned. It must therefore be supposed that some spasm, left after embolectomy, finally disappeared.

The second case is of a less familiar sort but as it demonstrates the good effect of resecting an artery thoroughly obstructed and irritated by an embolus, it is worth quoting

T H W, Surgical 50595, a man, fifty-six years of age, suffered twenty-four hours before presenting himself for treatment a tingling pain and pallor of his left hand. There had been no sign of heart disease. The hypothenar eminence of the hand was especially painful, the fourth and fifth fingers were "white as a sheet." Below the wrist, the whole left hand was cold, dirty white in color, the finger-nails dusky. The patient was so fearful of any contact that he guarded his left hand with his right. At first, a radial pulse was palpable but the blood pressure at the elbow was only 110 systolic as compared with a pressure of 170 on the right. A peculiar feature was a systolic bruit which could be heard just above the middle of the left clavicle.

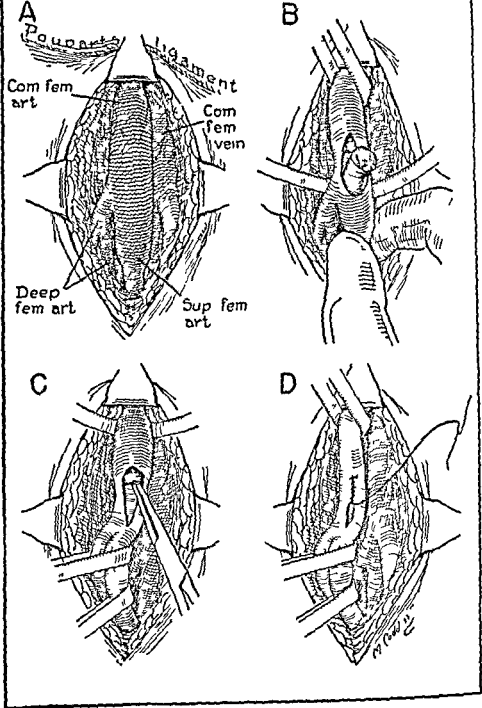
After nine days of treatment by intermittent use of alternate suction and pressure in the glass chamber, the bruit disappeared and a long tender mass could be felt in the lower course of the radial artery. The color of the hand as a whole had improved but the fourth and fifth fingers remained bluish white, clawed, and very sensitive to touch. The skin temperature of the left thumb and the tip of the little finger were barely lower than that of the corresponding parts of the right hand but rose only half as much as that of the right hand following a large injection of antityphoid vaccine. Some peripheral influence evidently was not only causing vasoconstriction but decidedly checking reactive hyperemia in the left hand. The tender, thickened radial artery, giving the impression of an acute arteritis, seemed to be responsible. It never occurred to anyone that an embolus had caught in the subclavian and later slipped down into the radial, yet such proved to be the case. Resection of the lower radial showed it to contain an embolus and at the moment of resection, under local anesthesia, the patient found himself suddenly relieved of his sense of coldness and sensitiveness in the fourth and fifth fingers. The color of the fingers improved and warmth

returned. The vicious circle of arterial irritation and vaso constriction had been broken up. Subsequently the state of the hand continued to improve.

*Treatment*—In spite of failures such as are illustrated by the case of F V D, failures which are only too common, embolectomy is still the method of choice in the treatment of embolism in the arteries leading to the legs. The Swedish surgeons, Key and Nystrom, have shown what can be done by concerted effort, but only when patients are promptly brought to the operating table. Unfortunately it is not easy to bring patients suffering from fibrillating hearts and suspected embolism quickly to the surgeon, nor is it always easy to be certain of a diagnosis, even when embolism occurs, as it is very likely to do, in a hospital. One can only hope that by education, better collaboration between physician and surgeon can be secured. It is especially important that hospitals should be equipped with the few simple tools necessary for opening and closing arteries. The fine needles and silk are now furnished prepared and sterilized. Soft rubber tubing is preferred for the purpose of lifting up and checking the flow in a large artery. A long, smooth probe, a fine rubber catheter, through which suction can be made, and one per cent sodium citrate solution or physiologic saline are readily available.

It has been abundantly demonstrated that after more than ten hours an embolus can rarely be successfully removed, because it will have so injured the intima that thrombosis will follow embolectomy, or thrombosis will have spread from the embolus into the peripheral tree. Even a four hour interval may be too long, yet one hears now and then of success after twenty four hours.

For the lower limb, attack on most emboli can be made from below the inguinal ligament. The most frequent site is the femoral bifurcation, yet if the embolus is lodged higher, it can still be dislodged by probing upward and by making an extraperitoneal approach through which extraction of the embolus can be aided by massage (Nystrom). By laying bare the common and superficial femoral arteries, through a generous



**FIGURE 12 ARTERIAL EMBOLECTOMY** A diagrammatic sketch after an actual operation A The right common and superficial femoral artery are distended by a great embolus B The femoral and its branches have fully been cleaned and isolated and soft tubing has been passed about them The lower part of the thrombus in the superficial femoral has been milked out C Retrograde bleeding controlled by tubing about the superficial and deep femoral branches While the common femoral is ready to be compressed at any moment by the upper tubing the upper part of the embolus is loosened D All bleeding controlled Repair, evertting the intima

incision made under novocaine infiltration, the site of the lesion can almost certainly be discovered and further exploration, if necessary, planned. The artery must be handled as little as possible, its intima touched only with the smoothest probe or blunt hook. Suction through a soft catheter and gentle pressure will deliver most emboli once they are loosened. In closing the artery, intima is united to intima with a continuous stitch of fine Chinese silk.

For the arm, embolectomy is seldom required, yet it is, if properly performed, all gain and no loss and may well be attempted in elderly persons in whom a good collateral circulation is unlikely to develop.

For long standing cases, in which there still seems some hope of saving a limb, resection of the plugged artery offers a decided advantage. For it often relieves vasospasm in the peripheral part of the arterial tree, and may just turn the scale in favor of a nearly gangrenous foot or hand.

*Conservative Treatment* should invariably be used (1) at an early stage until operative treatment can be secured and (2) when embolectomy is for any reason impossible of accomplishment. It consists in the application of warmth to the body and to the affected limb, slight lowering of the limb, between ten and fifteen degrees below the horizontal, and the injection of such a vasodilator as papaverine hydrochloride. Such measures are fully discussed by Allen and by De Takats.

Heat is applied by placing a large cradle over the lower half of the patient's body and legs. The temperature should not exceed 100° F (38° C). Such heat, by releasing vasoconstriction, secures the maximum dilatation possible. The leg had better, to conserve its heat and because warming the body under the heated cradle is preferable to heating the limb itself, be wrapped in woolen coverings.

Lowering the limb makes entry of the arterial blood easy, but the color of the skin must be studied. The toes (or fingers) should not be made too pale or too cyanotic but, if possible, pink.

Papaverine hydrochloride is given intravenously in a dose



of one-fourth grain (0.015 gm.) dissolved in salt solution. Twice this amount can probably be used with safety and the dose can be repeated.

Whether suction and pressure or intermittent venous compression shall be used depends upon whether such apparatus is easily available. There is a general feeling that this treatment is unlikely to save a limb which is not benefited by the measures already described. It is more likely to help develop a collateral circulation once the immediate crisis is successfully passed.

### JUVENILE GANGRENE

This rare and little understood form of gangrene occurs in children. Almost any pointed or terminal parts, usually many simultaneously, become necrotic—the nose, ears, toes, fingers, the tips of the elbows, the knee caps. But gangrene may also involve a whole limb, or several limbs. As a rule, the gangrenous part dries, shrinks and turns black. The background is usually an acute febrile infection, occasionally a debilitating illness.

From Martin's excellent account of four cases and his study of the literature, one gets the impression that bacteria carried into the blood stream may occasionally attack the wall of one or more large or many small arteries. The vessels are sometimes plugged by emboli, sometimes thrombosed, sometimes the seat of arteritis without thrombosis, and on occasion are undoubtedly thrown into a state of violent spasm by thrombosis of their companion veins, a matter more fully dealt with under diseases of the veins. It is hardly credible that any peripheral arterial thrombosis or gross embolism in a child can of itself make an obstruction sufficient to cause gangrene, as of a whole limb. There must be, in all cases, an element of secondary vasospasm. But this vasospasm, if present, is a single episode, and not in any sense recurring, as in the Raynaud's disease or the reactions to cold. It must then be supposed that juvenile gangrene can arise under a variety of circumstances utterly unpredictable and generally in the pres-

ence of infection, that the arterial or venous occlusion which occasions it is a source of such local and prolonged vasospasm that considerable masses of tissue may undergo rapid necrosis, and that repetition of the episode need not occur, provided the basic disease is relieved.

*Treatment*, being powerless to prevent gangrene, is devoted to curing the infection back of it if such is discovered. That, however, is routine, as in the case of diphtheria, typhoid, or pneumonia. If the child is debilitated and anemic, transfusions are apt to be useful. The gangrenous part must be allowed to separate, the adjacent tissues being encouraged to heal, so that every living bit can later be used to secure, by a plastic operation, the most useful amputation stump, or the least disfiguring remains of an ear or nose.

#### ERYTHROVELALGIA ERYTHERMALGIA

This peculiar and rare symptom complex is the very opposite of Raynaud's vasoconstriction in response to cold. It is, in fact, a vasodilatation in response to heat. As Weir Mitchell first described it, the symptoms appear in middle life. The individual first notices pain in the ball of the foot or toes upon standing, walking, or even letting the legs hang. Soon the dependent part becomes deep red and sensitive to pressure whenever the pain appears. With the deep redness go engorged veins, pulsating arteries and a hot skin. Since that early description, the disease has seemed rather vague and difficult to identify. Most physicians go through life without ever seeing a case, and Smith and Allen, in a recent paper, present only five cases from the Mayo Clinic.

The original name, of Greek derivation, meant red extremity pain. Smith and Allen suggest the name "erythromalgia", meaning red heat pain. They find that the individuals who present the very peculiar symptom complex are sensitive to heat, that is, in one or more limbs—the feet, hands, or both parts. Once the hand or foot has undergone the violent vasodilatation, the temperature of its skin is found to be elevated to the limit. But the bouts of vasodilatation continue

unchanged for so long as the warmth is maintained. Heat the body enough to cause even a slight reflex hyperemia, and as the skin temperature of the part rises to  $32^{\circ}\text{C}$  ( $90^{\circ}\text{F}$ ), vasodilatation is so accelerated that a temperature of nearly  $36^{\circ}\text{C}$  ( $97^{\circ}\text{F}$ ) is soon reached. Thus  $32^{\circ}\text{C}$ —the point varies slightly from person to person—is a critical level, a sort of trigger point. As long as the skin temperature remains in the sensitive range, between  $32^{\circ}$  to  $36^{\circ}\text{C}$ , the flushing, heat and pain continue. The discomfort can even be brought on by artificially inducing venous congestion when the critical level has not quite been reached.

The pain comes on as a tingling or pricking, not well localized, but once the hyperemia is full blown, the ball of the foot and tips of the toes, or the corresponding parts of the hand, suffer the unpleasant burning sensation, like a severe sun burn, which Mitchell described. To ward off the painful flush, the individual does not hesitate to expose the part to cold, sometimes sleeping with the feet out of bed, going about without shoes on cold floors, and, of course, elevating the feet. When the hands or feet are not flushed, there is nothing to observe, though vasoconstriction is sometimes present. Polycythemia has been noted in one or two cases.

*Treatment*—Aside from the measures instinctively taken by the resourceful individual to keep the extremities cool, there is little to be done. One of Smith and Allen's patients obtained considerable relief from 0.6 grain of acetylsalicylic acid but why the salicylates should be useful is unknown. Local nerve divisions have been tried without much success.

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## CHAPTER V

### VARICOSE VEINS

VARICOSE veins are those which have lost the power to transmit blood toward the heart against gravity. They are usually dilated, tortuous, and fibrosed. Above all, the cup like valves, indispensable to their normal function, are disabled. Varicosity is practically confined to the lower extremity and, for reasons which will presently appear, to the parts superficial to the muscular aponeurosis.

Varicose veins are totally useless. When the body is erect, blood actually flows down them and must be carried off by alternate routes. The very fact that in their presence venous blood is still able to return from foot to heart against gravity guarantees the efficiency of those alternate routes. Thus, their removal or destruction must always benefit and can never harm the venous circulation. For if the efficient veins are able to carry, in addition to their normal load, the down flowing blood of the varicose vein, all the more easily will they function once this unnatural load is removed.

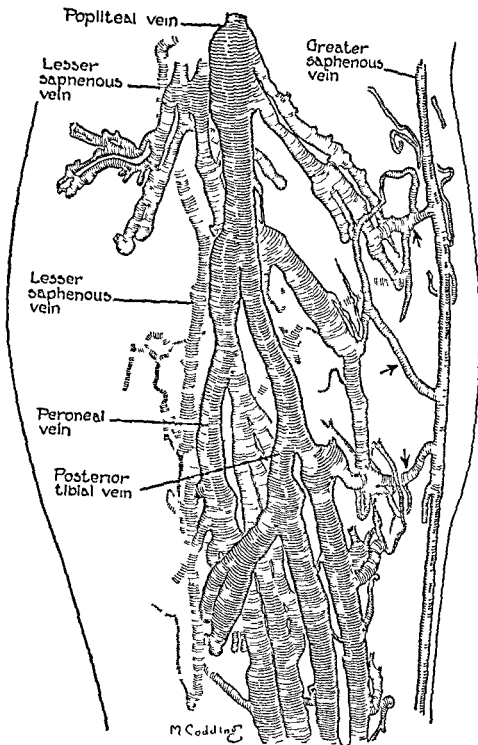
Varicose veins, in the dependent position rid themselves of their contents by the aid of two sorts of vessels, namely, the communicating or perforating veins, which pass the stagnant or downward flowing blood through the muscular aponeurosis into the deep system, and the deep veins, which then carry the blood up the leg to the body. Thus the communicating veins are a safety vent for the superficial vessels, but the deep veins actually do the work. Should the capacious, deep vessels find this something of a burden, the legs feel heavy under exertion and tire easily. But should they actually break down, the feet must turn purple and swell. Such an event is extraordinarily rare. Absence of blueness in the dependent foot of a person suffering from varix is sufficient proof that

the deep veins are functioning normally. If the above is true, some of the modern ritual of tests for varicosity is needless. These categorical statements have an anatomical, physiological, and pathological background which is offered in the following paragraphs.

**The Muscular and Valvular Mechanism Governing the Forwarding of Venous Blood from the Legs**—Blood is pushed into the veins of the legs under very low pressure from the capillary bed, the strength of the arterial stream being nearly lost and, when the individual stands, must force its way against gravity to reach the heart. Suction from the thorax can hardly be expected to do more than draw blood toward it from the great abdominal veins. The ability of the legs to empty themselves upwards, under these conditions, is due to their muscular quality and the presence of valves in the veins. The veins are enclosed in what amounts to a muscular envelope, and the valves, bicuspid as a rule, are so placed that blood can flow past them toward the heart but never back. In effect, they divide each vein into a long series of segments in such a way that muscular pressure, intermittently applied, permits them to be filled from below and emptied upward.

Not all the veins of the legs are equally exposed to this favorable action. The deep ones, enormously greater in number and capacity, being enclosed within the muscular aponeurosis, are always supported by the tonicity of the great muscles of the calf and thigh, and rhythmic motions, such as walking or running, empty them very effectively. The superficial veins are less favorably placed. They lie embedded in fat between the elastic skin and the fascial envelope of the muscles. Muscular pressure upon them is therefore indirect and is greatly dependent upon the resiliency of the superficial tissues. As years go by and the skin loses its elasticity, the subcutaneous tissue its firmness, muscular pressure becomes less and less effective. Thus the deep veins, well protected at all times, continue to function normally but the superficial veins tend to become distended, to lose a set of valves here and there, and if additional strains are put upon them, fail at last.





**FIGURE 13 THE VEINS OF THE LOWER LEG** Sketch after a roentgenogram of an injected leg (presumably at autopsy) Notice the enormously greater capacity of the deep as compared with the superficial veins Arrows point to some of the communicating veins The bones have been removed (Sketch made after Charles Remys "Traité des Varices", Figure 8 Courtesy of Vigot Freres, Paris )

to transmit blood against gravity These strains are various and will presently be discussed. The valves require immediate consideration

The valves are distributed at such intervals that in the great saphenous vein, which drains the median face of the calf and thigh, there are more than a dozen between the foot and groin There is also one in every entering branch, close to the parent stem In the perforating veins, which communicate between the superficial veins and the deep system, the valves are usually so set as to allow blood to flow inward but not outward However, in all communications of this sort the valves permit the most ingenious alternate routes In time of need, when one part or another of the venous system is obstructed, venous blood is able to pass in a direction never normally taken. The uppermost valve in the veins of the legs is found, rather inconstantly, in the external iliac

The local arrangement of the valves, as Edwards has shown, is such as to insure their perfect action when muscular pressure is applied to them Where each pair is attached, the vein is slightly elliptical in cross section, the major axis of this ellipse being parallel to the overlying skin, and each cusp arising from a long side of the ellipse Thus the crack between the opposing edges of the cusps is likewise parallel to the surface of the limb, and as the skin, or the underlying aponeurosis, presses upon the vein, the latter is flattened and the edges of the cusps are brought together Blood can of course flow upward past them but is less than ever able to flow back. When only a large single cusp is present, its situation is similarly favorable Occasionally three cusps are found Naturally, the valves are dependent upon a healthy state of the vein's wall They may lose and again regain their competence as the vein becomes overdistended and returns to its natural size But permanent distention and fibrosis leave them functionless and they are unable to resume their effective shape after thrombophlebitis

**Etiologic Factors in Varix.**—Varicosity of veins is perhaps most often due to increased intra abdominal tension such as

is caused by heavy labor. Long hours of standing predispose to it. So does pregnancy, but just in what way is not clear, for the superficial veins of the legs may be uncomfortably dilated as early as the second month of pregnancy when increased abdominal tension would seem not to be a factor. In other cases, the veins only become distended late in pregnancy, and not until several children have been born is varicosity established. Young persons of either sex, girls as a rule, occasionally begin to notice varicosity at about the time of puberty, after which the condition becomes progressive. Here there appears to be an inborn defect, of valves perhaps, which leads to dilatation of the superficial veins when the legs undergo rapid elongation. To some, this type suggests an endocrine influence.

Thrombophlebitis occasionally leads to varicosity. When, as occasionally happens, the superficial veins, especially those of the groin and thigh, become engorged as collaterals during obstruction of the external iliac and upper femoral, they are apt to remain dilated and so a completely varicose superficial system results. In other cases, the previously normal great saphenous itself becomes thrombosed either independently or in association with a femoral thrombophlebitis. Then, its valves being destroyed, it loses its power to forward blood against gravity, though it may remain a small, firm, straight cord.

**Anatomical and Pathological Features of Varix**—It is the great saphenous vein and its branches, the principal drainage system of the front and median face of the thigh and leg, which habitually become varicose. The lesser saphenous is sometimes involved through its connections with the greater but may become varicose when the saphena magna is altogether normal. In the latter case, dilated veins are evident upon the back of the calf and external surface of the ankle, close to the heel. When the lesser saphenous shares varicosity with the greater, it serves as a leaking communicating vein, as will appear below in the description of the tests for varicosity of the superficial and communicating systems.

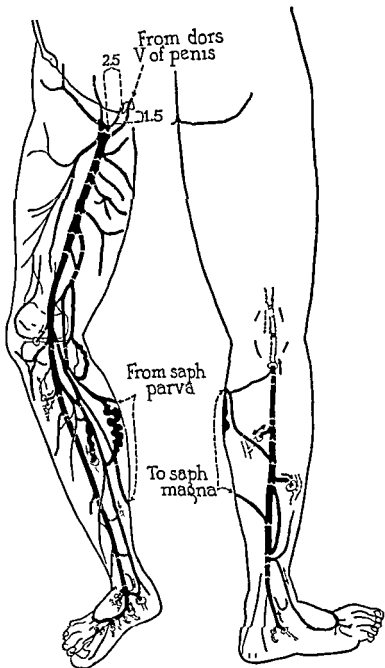


FIGURE 14 THE GREAT AND LESSER SAPHENOUS SYSTEMS OF VEINS their connections and a few of the communicating veins Note the many branches of the great saphenous close to its entry into the femoral In the female, some of these would run to the vulva. The valves are accurately shown From a dissection. Reproduced from Edwards, E. A., 'The Treatment of Varicose

The great saphenous vein, as the sketch shows, enters the femoral at the saphenous opening. Here it is joined by various superficial branches, some of which come from the lower abdominal wall, some from the pubic region, and some from the thigh itself. These vessels must carefully be divided when the saphenous is resected at the saphenous opening. For if a varicose stump is left, and these little veins with it, a new varicose system, surprisingly like the old one, is soon organized. The great saphenous occasionally splits, in the upper thigh, into two vessels of nearly equal size, but the main stem will always be found to pass toward the median posterior face of the knee. Just below this point a rather constant diagonal branch comes off and slants across the shin to the outer face of the calf. The principal vein heads straight for the internal malleolus. It is in connection with these vessels of the calf that the principal lines of perforating or communicating veins, of which Linton has recently given so full and accurate a description, are found. The communicating veins of the thigh are few in number, inconstant, and of little clinical importance.

The onset of varicosity is usually consistent with the notion that back pressure causes the saphenous vein to dilate and its valvular mechanism to fail, that is, that varicosity travels from above downward. For, though dilated, tortuous veins are first noticeable in the calf, examination of the groin will often show, at the same time, a full, tense, dilated area over the saphenous opening. In thin men, such an area forms a visible, rounded lump which transmits an impulse on coughing or straining and is easily mistaken for a femoral hernia. In women, the subcutaneous fat is so thick that the dilated saphenous vein of the thigh is seldom visible, though it can usually be traced upward from below by palpation. The fact is that in almost every case where permanently dilated veins are visible below the knee, a flow of blood down the great saphenous is obvious, as evidence that the valves in the thigh are gone. Sometimes a down flow can only be demonstrated when the individual has long been on his feet, suggesting that some

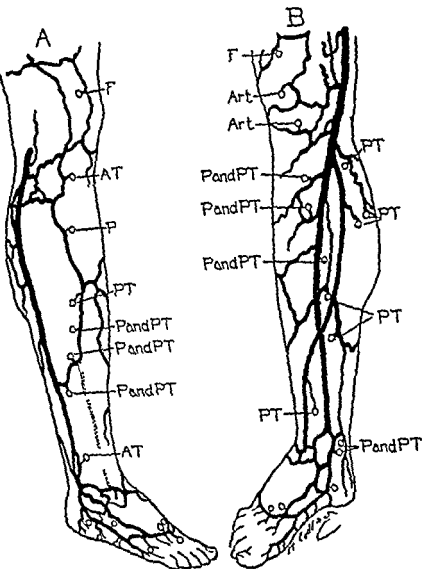


FIGURE 10 THE GREATER, A, AND LESSER, B SAPHENOUS SYSTEMS OF THE LEG, showing their communicating branches after Charles Remy (Courtesy of Vigot Frères Paris)

The communicating veins are shown by circles and their connections with the deeper vessels (according to Remy) are indicated by letters as follows *F* femoral *Art* articular *AT* anterior tibial, *P*, perineal, *PT* posterior tibial. It would seem that the communicating veins in *A* marked *P* and *PT*, should naturally join the anterior tibial or perineal veins, and that those in *B* marked *P* and *PT*, should join the posterior tibial exclusively. In any case, this sketch gives a good idea of the number and distribution of these vessels.

valves still function for a time after the vein has, by elevation, recovered its lost tone. There is, however, such a thing as local varicosity, if one so chooses to name it. That is, a stretch of some visible superficial vein is thin walled and snake-like though never greatly dilated, yet no general varicosity of the great saphenous system is evident. Such a state is occasionally seen in multiparous, adipose women and were it not that a thrombophlebitis sometimes starts in the locally abnormal vein, would be of little consequence.

The pathological change in the wall of the varicose vein is one of fibrosis. At first, the vein, though abnormally distensible, is still elastic and capable of contracting. Later it becomes permanently dilated, tortuous, and finally hard, even calcified. It often gives way here and there, making pockets, some of which reach considerable size but chiefly it takes on a writhing snake like appearance, projecting above the skin in a very obvious manner. In thin persons, such a state is almost unmistakable but in the adipose, the skin may be smooth and the dilated state of the veins hardly visible. In some cases, the main channel of the great saphenous vein is a single, grossly distended, tortuous cord. The branches of such a vein are still quite normal, and the blood it receives is carried off by the communicating vessels. In other cases, there are several large varicose branches both in the thigh and calf, but the continuity of the whole venous tree is evident. The capacity of such a system is considerable, and one would suppose that the patient, on getting up in the morning, would lose so much blood into it as to become faint, yet one never hears this story. Rarely, varicose veins are extraordinarily diffuse, coming to the surface here and there rather than continuously, all over the leg.

The state of the skin in most cases of varix is not remarkable. Occasionally, pigmented areas appear upon the lower third of the leg, but no sign of malnutrition need be present. Certainly, varicose veins lead to no serious degree of edema or cyanosis. In other words, the collateral circulation which takes the place of the great saphenous system is usually effi

ment. Ulcers appear to be due to injury, local malnutrition, and infection. They will be considered in a later section. It is astonishing how nearly their location corresponds to that of

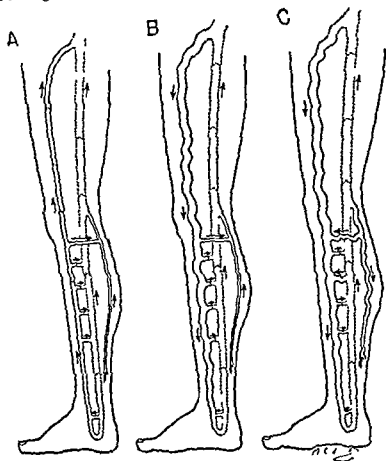


FIGURE 16 A The direction of the current in the normal superficial (solid lines) and deep (dotted lines) veins B Superficial Varicosity The communicating veins, including the lesser saphenous are competent and act as safety vents for the varicose veins C Superficial Varicosity with incompetence of some of the communicating veins, among them, the lesser saphenous.

other ulcers of the leg, that is, in the lower third, more often on the median side than elsewhere, seldom below the ankle, but if so in the region of either malleolus and never on the foot proper. They tend to ride upon a varicose vein. Even when



the area of the ulcer is greatly indurated, a varicose vein can usually be traced down to the site of the sore

*The Subjective Symptoms of varix* are often remarkably slight. It seems to cause more discomfort in the way of tingling or aching when partly developed than when full blown. Indeed, well compensated varicosity would be ignored more often than it is if it were not so unsightly, but when varicose veins do cause symptoms the leg is apt to feel heavy on long standing. The skin may tingle and often itch quite severely. All such troubles are confined to the lower leg as if the degree of back pressure was responsible for them. The large veins of the thigh are seldom a source of discomfort, though occasionally, in adipose women, a very tortuous, distensible, superficial vein in this region causes pain. Ulceration and thrombosis, after all, chiefly cause the varicose individual to complain.

**The Diagnosis of Varix** **Trendelenburg's Tests**—Trendelenburg had a very profound understanding of varicose veins, and the simple examinations he devised still afford all the information which anyone requires for diagnosis. He showed that varicose veins, once emptied of blood by elevation, fill by a downward rush of blood on depression. He understood how the communicating veins are able to carry into the deep system the blood accumulated in the functionless superficial veins. The valves of the perforators, he realized, are so set as to favor this flow, but he pointed out that, even when no valves are present, once pressure rises higher in the long varicose column than in the well valved deep vessels, blood must pass from the former into the latter. Finally, he devised a test for the efficiency of the communicating veins.

Trendelenburg's first test consisted in emptying the varicose veins by elevation, after which the leg was depressed and the patient stood up. The blood can usually be seen to flow into the large veins, distending them rapidly from above downward. Let this be called a positive Trendelenburg test. However, the test is not always easy to carry out, nor is a complete absence of valves invariably evident. Varicose veins are often so little visible, or it may be so hard, that the down flow

can not be seen It can then be detected only by placing the finger tips upon the empty veins, at a point just below the knee, and noticing their tension as they fill from above To carry out the test, then, begin by having the patient stand, and view the whole limb, noticing the course and prominence of the veins Decide whether the down flow of blood shall be detected by sight or palpation, seat the patient in a very strong chair, and let an assistant tip the chair back As this is done, elevate the leg to be examined, inspecting, and palpating its empty vessels Then let the chair be tipped forward and let the patient stand Should the veins fill with a shock, varicosity is evident enough If they only fill in five to ten seconds, some valves are still present and are able, until distension of the vessel is complete, to delay the downward flow Such an event is of no great importance, varicosity may be counted present

That blood actually flows down a valveless vein when the leg is dependent requires little proof Without valves, blood could never mount against gravity But actual proof has been secured by McPheeters and others that blood descends a varicose vein, passes through communicating vessels into the deep system, and there mounts toward the heart That is, opaque material has been injected into a varicose vein and traced by the aid of the X ray along the course described above

Next comes the *Trendelenburg Test with Constriction* Blood is known to flow down the valveless vein The question then is can blood still flow into the varicose veins of the lower leg when prevented from flowing down from above? In fact, do the communicating veins leak, allowing blood to escape from the deep into the superficial system? To test this point, the patient is again seated in a chair, and tipped back, the leg raised and emptied of blood. But now carry a piece of bandage about the mid thigh, passing its ends through the fingers just as reins are, or used to be, held in driving a horse A twist of the hand tightens the bandage and the patient stands Again the varicose veins below the knee must be palpated if their state of emptiness or distension is not plainly visible The constriction indents the thigh sufficiently to prevent a

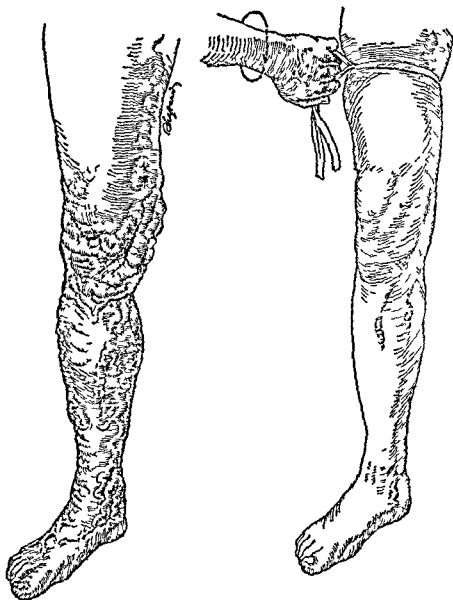


FIGURE 17 THE TRENDLENBURG TEST, WITH CONSTRICTION The leg, having been emptied of blood by elevation, is subjected to constriction in the mid thigh by the tightening of a piece of bandage In purely superficial varicosity, down flow of blood is prevented and the superficial veins, below the constriction, do not fill—for half a minute or more Even when moderately full, they become increasingly tense on release of the constriction In the presence of incompetent communicating veins, filling occurs rapidly below the constriction and there is little or no additional impulse on release of the bandage (From Homans's *Textbook of Surgery* Courtesy of C C Thomas, Springfield, Ill , and Baltimore, Md )

downward flow in the varicose veins but does not interfere \* with the circulation beneath the muscular aponeurosis. Should the varicose veins remain empty and relaxed below the constriction for half a minute to a minute (depending upon their capacity, for they must finally be filled from the arterial side) the test may be considered negative, that is, the communicating veins are competent and no blood escapes through them from the deep to the superficial system. Thus being determined, what the French have called the "contre épreuve" of the Trendelenburg test may now be completed: the bandage is released and the impact of the down flowing blood into the varices below is seen or felt.

A positive constriction test leads to further discoveries. Suppose the varicose veins fill below the constriction in five, ten, or fifteen seconds, proving that some communicating veins are incompetent. It may be desirable, especially if a varicose ulcer is present, to discover at about what level the leak or leaks occur. A varicose lesser saphenous vein, which has connections with the greater and empties into the popliteal, should first be suspected. So apply the constriction to the elevated leg just below the patella and repeat the test. If the veins now fail to fill below the constriction the problem is solved. The lesser saphenous vein is almost certainly varicose and connects with the varicose great saphenous system—blood leaks out through it from the popliteal into the superficial veins. Therefore, to free the superficial parts from venous stasis the lesser as well as the greater saphenous vein must be divided, obliterated, or removed. Beyond this it is not easy to go. If the superficial veins still fill, on depressing the leg, when the constriction is applied at the knee, it is only possible to say that incompetent perforators are present at a still lower level.

There is a good reason for using a bandage rather than a piece of rubber tubing. The latter is so yielding that it fails until drawn very tight to shut off completely the downward flow in the superficial veins and when drawn tight is very likely to interfere with the venous return beneath the aponeurosis making the foot cyanotic. Whereas it is very easy to twist the bandage just tightly enough to prevent any flow down the varicose veins without causing any deep venous congestion whatever.

However, one may sometimes learn a little more. One may wish to know, for instance, where most of the communicating veins are situated. So the leg is depressed until the varicose veins are full and tense. Then, with the finger tips, one compresses the great saphenous at the groin and elevates the leg, say to an angle of  $20^{\circ}$  above the horizontal. As a rule, the veins of the thigh remain full, there being no perforators (or insignificant ones only) above the knee. Below the knee, the superficial veins will usually empty themselves through perforating veins into the deep system, so that one can detect the level of the highest (cephalad) of the latter. One can then, with one's free hand, sweep the blood from the thigh toward the foot and note at what point most of it disappears from the surface vessels. In the jargon of varix, a positive constriction test—filling of a varicose vein below the constriction—is called a "Trendelenburg double"

*The Schwartz Test*—In 1908, Chevrier published a monograph on varix in which he called attention to the "Signe de la Chiquenaude de Schwartz." With the leg horizontal, the full vein was tapped in the thigh and the consequent undulation traced toward the periphery, thus demonstrating the absence of valves. The writer (1916-27) having adopted this test to his own purposes, used it in the erect position, making the wave travel from below upward and using it to trace the course of the varicose vein in the adipose thigh. To this end, the varicose vein below the knee is snapped with the back of the finger-tip and the shock is felt by the flat of the fingers of the other hand placed upon the inner face of the thigh. Only a varicose vein will transmit such an impulse. Thus the sign is in a way a test for varicosity.

Others have observed the same phenomenon.

*The Perthes Test*—A number of clinicians, whose opinion deserves respect, make use of this test. Its object is to prove, or disprove, that in the presence of superficial varicosity the deep system of veins is functioning normally. Perthes noticed that after Trendelenburg's division of the varicose saphenous vein in the thigh or when the vein was compressed high with

the fingers, exercise caused the calf to become smaller, by which he judged that the pumping action of the muscles was emptying the leg of blood effectively through the femoral (deep) system. On the other hand, if the blood were free to pour down the varicose vein, the calf failed to shrink and even became enlarged, the task of the communicating and deep veins being one of cleaning, as he said, the Augean stables! His compression of the great saphenous at the groin was actually a test before operation of how much more effectively the deep veins would empty the leg of blood after the down flow through the varicose saphenous vein was cut off than before. However, he did not propose high compression of the varicose great saphenous vein either as a test of obstruction or efficiency in the deep venous system. It is others, in subsequent years, who have held it to be available for this purpose, and recently Mahorner and Ochsner have used it to bring out some rather fine points in diagnosis. Tying a piece of rubber tubing about the thigh, they set the patient to walking and notice whether the varicose veins, below the constriction, shrink or remain full. If the veins shrink, the deep veins are effectively draining the varicose system and are themselves patent and efficient. By applying the elastic constriction at various levels in the thigh, they hold that they can identify a point at which the deep veins are obstructed or at which a leaking perforating vein is present. The test ignores the principle already laid down earlier in this chapter, namely, that merely walking or standing, with no constriction applied, tells the story of the deep veins in the presence of varix. For since the deep vessels must *always* do the work of the varicose veins, beside their own, their efficiency is already tested and proved if the foot of the varicose leg does not become cyanotic on walking and standing. One can compare the varicose leg with the normal one, or two varicose legs with those of an otherwise comparable individual. Finally, it should be pointed out that the right degree of constriction with a piece of rubber tubing—improperly called a tourniquet—is difficult to secure. In fact, the Perthes test, with its variation, is one for

experts Skillfully used, it secures information about the level of incompetent perforating veins The presence or absence of valvelessness or obstruction in the femoral system is evident without its aid

**Varicose Ulcer**—Ulcer is the most common complication of varix and is chiefly responsible for the disablements due to that disease It has already been explained that the varicose leg often presents, throughout life, a healthy skin The change most frequently seen is a deposit of brown pigment, first as a local speckling, later as a deep brown patch Such may precede ulceration The change seems to indicate malnutrition and to appear most often in the lower part of the leg, in front or upon the inner surface, occasionally over or below the malleoli

Ulcers are apt to appear in pigmented areas and on the course of veins, that is, they tend to "ride" veins A trifling injury usually initiates the process The first sore is trifling, shallow, and as a rule is readily healed by cleanliness and the use of a bandage sufficiently firm to compress the veins to which the ulcer is tributary But a spot once ulcerated, tends sooner or later to break down again In time, the sore enlarges and becomes the center of an area of more or less marked induration and edema Just how varicosity lowers the resistance of the tissues, is immaterial Local malnutrition, followed by injury and infection, is sufficient excuse In some instances, however, the location of a sore is dictated by a great lake of varicose vessels or by the presence of a leaking communicating vein beneath This latter possibility needs keeping in mind, for if a leaking perforator is present, merely doing away with the great saphenous vein proximal to the ulcer will rarely cure it, local venous stasis being maintained by the incompetent communicating vein This is the best reason for using the constriction test to detect the presence of such veins It may call for special operative treatment to rid the leg of them

Ulcers are more or less painful and disabling according as they are or are not inflamed But even a badly infected ulcer,

as indicated by the redness, swelling and tenderness about it, is more painful in certain situations than others. The worst are low down upon the inner face of the leg overlying the lateral malleolus. Perhaps they derive their painful quality from the presence, beneath them, of the long internal saphenous nerve, which is closely associated with the great sa

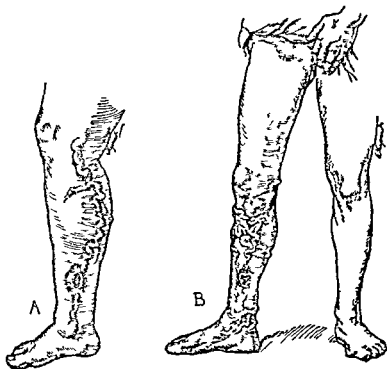


FIGURE 18 VARICOSE VEINS AND ULCERS A The ulcer 'rides' upon a vein B The ulcer is in the mid-t of a considerable induration and is less directly related to any one varicose vein. At the groin, a dilatation over the root of the great saphenous vein is shown. (From *Homans's Textbook of Surgery* Courtesy of C C Thomas, Springfield, Ill, and Baltimore, Md.)

phenous vein in the calf and supplies the skin of the median face of the leg and ankle. It will sometimes be found advantageous in treating the ulcer to divide this nerve near the knee.

The favorite situation of varicose ulcer does little to distinguish it from other sorts. The postphlebotic ulcer is most



often found in similar locations, and even the unexplained ulcers of the adipose are usually seen on the median face of the leg in its lower third. Nor does the appearance of a varicose ulcer mark it in any way. It is usually shallow and not undermining, but so are most other ulcers (except syphilitic ones). It rarely encircles the leg but others rarely do so either. In fact, a varicose ulcer is identified principally by a history of, and by the presence of, varicose veins. Destroy the varicose veins and some varicose ulcers will persist, either because of great thickening and scarring of the surrounding tissues or because of incompetent communicating veins beneath. But just because an ulcer *looks* like a varicose one, it should not so be labeled, and treated, unless varicose veins are plainly responsible. In these days, when a multitude of clinicians seem to be in the habit of filling any vein they can find with a sclerosing solution just because an ulcer is present in the lower leg, this prohibition is important. Many ulcers so treated are postphlebotic ones and are made worse by such thoughtless acts. The postphlebotic ulcer will subsequently be discussed as a separate problem.

**"Varicose Eczema"**—In a small percentage of cases, a patch of dermatitis, usually described as "eczema", is associated with varicose veins. This is most often situated on the inner face of the lower half of the leg, just as is the case with ulcer, but may appear upon the outer surface or even occupy the whole lower leg. The patch is slightly elevated, reddened, scaly, and constantly weeps, but is not actually ulcerated. Plate VI, facing page 202, illustrates an advanced but perfectly typical disease of this sort. Apparently varicose veins merely occasion the change which favors the establishment of such lesions, and it is almost equally probable that all these dermatoses, if such they may be called, are not of one sort. However, since signs of fungus infection on the feet—scaling skin, moist, itching patches between the toes—almost invariably are present, it may be supposed that most of them are allergic reactions to, if not the immediate seat of, epidermophytosis.

*Treatment* is exceedingly difficult. In the first place, the varicose veins had better be divided at the knee and groin. Even if incompetent perforating veins suggest the need of a dissection of the calf, this will hardly be possible in the presence of an infected skin. Probably life in bed for a week or more, while gentle liquid fungicides, such as potassium permanganate (1 to 2000-3000), thymol or salicylic acid in fifty per cent alcohol (1-2%) or watery aluminum acetate (1 to 12-20) are applied to the leg and foot, should precede the high low division of the saphenous vein. Any considerable dissection must be postponed till later.

In the subsequent treatment of the "eczema", the sensitive ness of the patient to fungi as well as other possible causes of an allergic reaction should be studied. Any recognizable epidermophytosis should be treated by fungicides carried in solution or ointment as trial directs.

#### TREATMENT OF VARICOSE VEINS AND ULCER

**Nonoperative Treatment.**—This comprises the application of bandages or stockings and the injection of sclerosing chemicals.

The principle upon which *Bandages* and *Stockings* are applied is a very simple one, namely, that the blood which otherwise would fill, distend, and even flow down varicose veins is by the pressure of the bandage prevented from so doing. The varicose veins are compressed, though one can hardly expect them to be kept altogether empty, and the venous blood, instead of remaining stagnant in the superficial parts, is assisted by the elastic pressure to enter the deep veins which are quite able to care for it. The modern semi elastic cotton bandage is very effective as is the elastic stocking, but an unyielding bandage, if carefully fitted, serves almost equally well and can be left on for many days. A lace up canvas stocking is also very useful.

In the presence of ulcer, such palliative treatment is intended (1) to keep stagnant blood away from the sore and (2) to aid in applying such local ointments or antiseptic solu

tions as will diminish infection and favor epithelization. When afflicted with large, infected, and deeply indurated ulcers, the patient had better be confined to bed, the leg slightly elevated. Hot saline or boracic dressings should then be applied. A dehydrating solution such as glycerine and saturated magnesium sulphate, if not too painful, may be of advantage. As edema diminishes, the ulcer will take on a bright color and skin will begin to cover it. Such treatment must usually precede one of the operative measures later to be described. Even if it induces healing of the ulcer it can not be expected to produce a permanent cure.

For ambulatory treatment of a difficult ulcer, the use of the rubber sponge, or "venous heart" as described by McPheeters, is very effective. "A good grade rubber bath sponge is selected of a size larger than the ulcer. Some soothing ointment is applied to the ulcer surface. Fluffy gauze dressings are applied and a few layers of sheet wadding. Over this the rubber sponge is applied and bound in place with a plain gauze bandage. \* \* \* Now apply the four inch ace cotton elastic bandage starting at the knee and going downward with a double figure of eight about the ankle." Walking is then encouraged as it tends to pump fluid out of the tissues.

Another excellent ambulatory dressing, seldom used because its application requires time and pains, is the Unna's paste\* stocking. This is indicated when there is little infection but the ulcer is resistant to epithelization. The application of the stocking requires some skill. The leg, after being elevated for half an hour, is painted thoroughly with the warm liquid paste. At once a single layer of narrow gauze bandage is applied from toes to knee, over skin and ulcer alike. Upon this a second painting is made. Then another layer of bandage. In this way three or four alternating layers of paste and gauze are applied until the bandage is fairly firm though still flexible. If the ulcer is relatively clean, such a boot is left on for even a couple of weeks, at the end of which time healing

\* To make Unna's paste mix zinc oxide 10 gm., gelatin 40 gm., glycerine 120 c.c.m., water, 150 ccm. heat in a water bath to liquefy before applying.

will often be complete. Or a window can be cut for dressings. As a substitute for this boot, a very useful procedure, in the case of a clean, shallow ulcer, is to cover it with strips of zinc oxide plaster, which half encircle the leg and make a little pressure upon the sore. Over this, even without an intervening dressing, a bandage (from toes to knee) is applied.

The nature of the local application seems on the whole the least important aspect of treatment. In other words, vaseline gauze is about as efficient as boracic ointment and boracic ointment almost as good as an ointment \* of oxyquinoline and scarlet red. Sometimes a sore can be treated effectively by regarding it as a burn and painting it with a dye such as gentian violet (2 per cent), to form a dry covering, or with a 10 per cent solution of silver nitrate.

*The Injection of Sclerosing Chemicals*—This method has now been long enough in general use so that its virtues and failings can be fairly well evaluated. It is not a cure all for the lazy surgeon to use. Indeed, the successful injection of sclerosing fluids into varicose veins calls for a *high degree of skill and pains*. It is probably true that as the great majority of surgeons practice injection, they fail to obliterate permanently more than a small percentage of the veins they treat, which is not to deny that such injection may here and there close a short length of vein. However, their injections, by at least temporarily obliterating one or more veins leading to an ulcer, will cause it to heal while the patient is ambulatory, a most important consideration for those who must work daily. Two or perhaps three serious indictments can be brought against the injection method. (1) In unpredictable instances, a disagreeable, wandering, obstinate sort of thrombo-phlebitis is set up, (2) recurrences are common and often so diffuse as to be difficult to treat by any method, and (3) it is not easy to avoid an occasional slough.

	Gms. or Ccm.
Oxyquinoline sulphate	0.6
Scharlach R ointment 5%	120
Liquid Petrolatum	15

The first two undesirable happenings just described can be avoided, or rather minimized, by combining with the injection a high division of the great saphenous vein, that is, at its junction with the femoral. A retrograde injection made during this procedure is seldom followed by any serious degree of soreness of the injected veins and the combination certainly lessens enormously (over pure injection) the number of recurrences. However, high division, except in very favorable cases (thin men) requires hospitalization for forty eight hours. It is not a routine procedure for the outpatient department or office.

To these general criticisms it should be added that very large veins are difficult to close by injection, and that injection is particularly ineffective in the presence of leaking communicating veins (doubly positive Trendelenburg test). In the treatment of postphlebitic ulcer, injections are utterly useless and even dangerous, not because they may not obliterate a dilated vein now and then, but because they aggravate the already inflamed state of the tissues, increasing disability and pain and even causing new ulcers to appear.

The indications for injection, pure and simple, that is, without high division, are, first, the treatment of varicose ulcer when the connection between vein and ulcer is clear, especially in working people and the aged (in the former it should, however, be followed up by more radical treatment) and second, the obliteration of unsightly but otherwise symptomless varices of moderate size.

High division plus retrograde or secondary injection is indicated in the treatment of any varices at any age, provided that incompetent communicating veins are not present (indication for open resection) or that a deep thrombophlebitis has not left behind postphlebitic indurations.

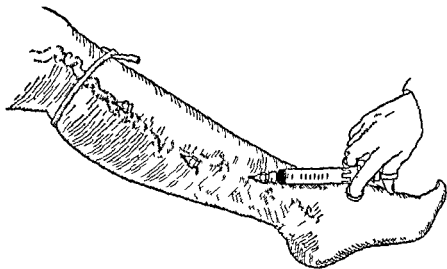
To the above should be added two general rules, which, if religiously followed, greatly favor the success of injection (1) Inject as great a length of vein as possible at one sitting, and (2) inject the vein when empty of blood, keep it empty for a few minutes afterward, and, by compression, prevent it

from being distended with blood during the following twenty four hours

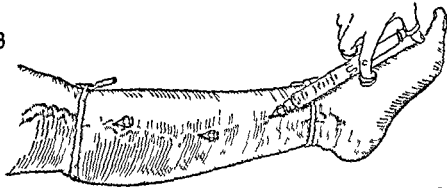
*Injection without high division*—The length of vein to be treated should carefully be studied. It may require the use of several needles, a matter calling for a certain degree of quickness and skill—one needle perhaps to a stretch of 7.5 cm. If the veins of both thigh and leg are to be treated, McPheeters recommends that the thigh be treated at the first sitting, the leg at the second. An encircling piece of rubber tubing prevents the sclerosing solution from passing down from the vessels of the thigh into those of the calf. The needles used should have a short bevel and should not be too fine lest blood clot in them before the injection can be made. Each is inserted attached to an empty syringe, while the vein is reasonably full of blood, the leg slightly dependent. Blood must then be demonstrated by suction, after which the syringe is detached and the needle, from which a little blood oozes, left in the vein. If only one injection is to be made, the leg is then raised and, to keep the vein empty of blood, a ring of lead wire is pressed down upon it, isolating the stretch of vein to be treated (or pieces of bandage or tubing are tied above and below the area). If several injections are to be made in series, an assistant may hold a piece of gauze about the open dripping base of each needle until the operator is ready to make his injection into them in turn. The injection is of course made when the leg is elevated. A long segment of vein can be isolated and kept empty by encircling rubber tubing.

For large injections, solutions of invert sugar (seventy five per cent), dextrose (fifty per cent), or sodium chloride (ten to twenty per cent) are suitable. Such are now available in sterile ampoules, also various favored combinations. As much as twenty ccm. of one of these solutions can be injected at one time. For injection into short stretches, a useful solution is quinine hydrochloride (gm. 0.26) and urethane (gm. 0.13) combined in a two ccm. ampoule. No more than this should be used at one sitting (vomiting may occur if the patient's tolerance for quinine is low). Another effective one is sodium

A



B



C

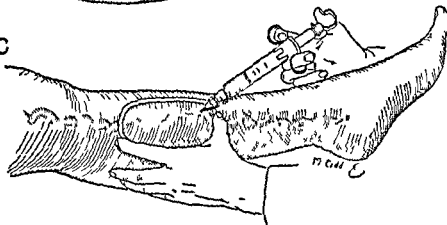


FIGURE 19 THE INJECTION OF VARICOSE VEINS A Needles are inserted while the leg is partly dependent and are left detached as successive ones are placed, a rubber band prevents down flow of blood in the vein B The leg is quickly raised to the horizontal and the injection made, between bands at knee and ankle, into empty vessels C The use of lead wire to localize an injection, as of quinine and urethane, or sodium morrhuate

morrhuate, in five per cent solution, of which two to five cc. may be used.

In former times, much sodium salicylate was injected in a thirty per cent solution (or even stronger). This, like strong sodium chloride, causes sufficient pain to call for a general anesthetic and is not now favored.

Following injection, the needle is left in place for a minute while the solution diffuses. As it is withdrawn, pressure is made over the spot with a piece of gauze. A pad is then applied over the injected area and held in place by a bandage. Undoubtedly the efficiency of the injection is increased if the leg is not lowered for twenty-four hours, though active exercise, immediately after the injection, has been advised to carry any excess of solution out of the deep veins. Just why this is proposed when moderate elevation drains the leg still more rapidly, is not clear to the writer.

*High Resection of the Great Saphenous Vein*—The idea of combining the injection of a sclerosing solution with ligation of the great saphenous vein in the thigh is an old one, and De Takats seems to have been the first to use a reasonably high division in ambulatory patients for this purpose. The writer prefers to speak of "resection" and that at the highest point possible, the saphenous opening. The word, "resection" is advisedly used in place of "ligation." A vein ligated in continuity re-establishes its channel with astonishing rapidity. Simple division between ligatures is hardly more effective. But division of the varicose great saphenous vein at its junction with the femoral, leaving no varicose stump and removing several centimeters of the vein below, is reasonably effective. Particular attention should be given to the branches entering the vein near its root. As these are divided they should be followed gently into the fat with the point of a fine hemostat and there ligated. It is easy to understand that if a varicose stump is left, with branches emptying into it, connections are soon made with neighboring veins and a new varicose system is soon established. To bear out and expand this statement, the writer has observed that among women



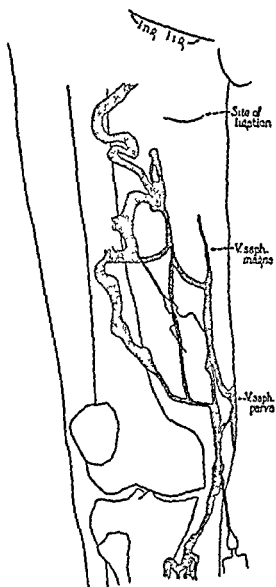


FIGURE 20 RECURRENCE OF VARICOSITY AFTER AN INSUFFICIENTLY HIGH SAPHEOUS DIVISION The new varicose connection might well have been shown passing into the stump of the great saphenous or the vulval veins After an actual dissection of Dr E A Edwards, who has kindly allowed the use of his sketch (Courtesy of *Surgery, Gynecology and Obstetrics*, 59 916 928 (Dec ) 1934 )

who have borne children and in whom large vulval vessels connecting with veins in the upper thigh are evident, it is a practical impossibility, by any method, to prevent a recurrence of varicose veins. One may resect the upper great saphenous with its entering branches, carry the incision medially close to the vulva, dividing all veins encountered, and make a retrograde injection of the main stem of the great saphenous (or even remove it down to the knee), yet, as a rule, a new set of varicose veins will be established within six months to a year. The explanation seems to be that many small potentially dilatable veins cross the operative field. Mere division of these fails to prevent their making some sort of connection across or around the reunited surfaces. Back pressure from above, there being no valves or only useless valves in the neighboring veins, soon opens up a new varicose pathway.

The technique of resecting the saphena magna at the groin is sufficiently illustrated in the accompanying sketches. The operation is performed under procaine infiltration. The one per cent solution is generously infiltrated into the skin, subcutaneous tissue, and, if the patient is not so fat as to make the whereabouts of the needle point uncertain, into the fascia lata. The operator then waits twenty minutes (by the clock). The vein is necessarily encountered by the oblique cut and once found is then and there isolated and divided between hemostats. It is then dissected upward until the operator can see the bend of the vessel as it enters the femoral, which he must be careful not to draw out. In this part of the procedure he will sometimes be aware of a vague group of lymph nodes, which may take the form of a faintly inflammatory thickening, rather lateral to the varicose stump. He should disturb these as little as possible or an occasional reaction will result, causing some local induration and swelling in the region of the wound. As the stump is lifted up, its entering branches are clamped, followed into the fat and tied with fine silk. Clamps on the stump's side are rarely needed. As the great saphenous itself is ligated (with larger silk), tension on the stump is released.

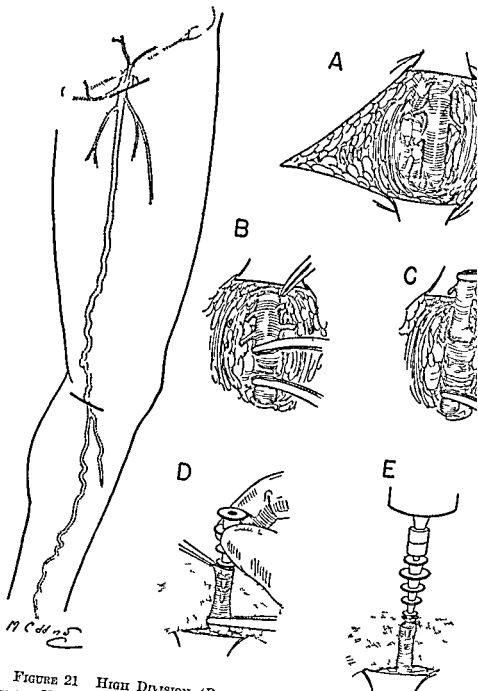


FIGURE 21 HIGH DIVISION (RESECTION) OF THE VARICOSE GREAT SAPHENOUS VEIN The low incision for the 'High Low Division' is also indicated  
 A The great saphenous and its highest branches B The varicose vein is divided well below the saphenous opening Its branches are followed into the fat, divided and ligated with fine silk. C The stump is turned up, ready to tie at its entry into the femoral D and E The insertion of the Mass Gen. Hospital "pagoda" into the distal stump, ready for injection of the sclerosing solution

to make certain that the femoral is not being drawn out and included in the ligature. A long cuff (15 cm. or half an inch) is left distal to the tie. The pessimist will apply two ties.

If a retrograde injection is to be made, it will be of advantage to have operated with the lower end of the table elevated perhaps six inches above the head. The veins will then be empty. By tying a cannula, which fits a twenty ccm. syringe, into the lower end of the divided vein, the fluid is easily injected without leakage. Any of the concentrated sugar solutions, combined with twenty per cent saline, can be used in an amount up to twenty ccm., depending upon the size of the varicose veins. There will often be a momentary cramp-like pain and, after the lower stump has been ligated with silk, the wound is irrigated with warm physiologic saline and closed with fine silk stitches so placed as to obliterate all dead space. A small local flexible dressing is then sufficient for the wound, but a gauze pad may be applied to the inner face of the thigh (to compress the injected veins) and held in place by adhesive strips or a bandage. How long the patient shall remain in bed depends upon the healing of the wound. The leg can be moved freely from the start. If the wound is reactionless one or at the most three days in bed is sufficient, but during this time the patient should remain prone, not sit up or recline. Whether he can then get about without discomfort will depend upon the reaction in the varicose veins. As a rule, a reasonably normal life can at once be resumed. For a reliable account of the ultimate success of this procedure, especially as compared with injection alone, Faxon and Barrow's paper from the circulatory clinic at the Massachusetts General Hospital should be consulted.

*Combined High-Low Resection.*—An advantageous variation upon high resection with retrograde injection, especially if the varicose veins are large and present one main channel at the knee, is to begin by dividing this vein—the foot of the table being raised six inches above the head—through a transverse incision just below (or above) the knee joint. In that case, any branch connecting with the lesser saphenous vein

can be cut off and the solution, later to be injected from above, will not run down and set up a useless, annoying secondary thrombosis in the calf. Needless to say, the division and ligation of all veins encountered must be painstaking and complete, lest a leak of the sclerosing solution into the tissues occur. Following this operation, and at the same sitting, the resection at the groin and retrograde injection are carried out. The high-low procedure is probably more effective than high resection alone.

*Operative Resection of Varicose Veins*—This operation, nearly discarded at one time in favor of injection, has a place under certain conditions. That it was a cause, in persons over fifty-five years of age, of just as much fatal pulmonary embolism as occurs in such individuals following *any* operation upon the abdomen or below is reasonably certain. That it was a cause of embolism, in younger persons, unless carelessly performed, is extremely doubtful. It may, in fact, be undertaken without hesitation, provided the operator is not planning to hurry over it, in any one, preferably in the twenties or thirties, who desires a radical cure of his varicosity. However, it is distinctly indicated (1) when, in a young or middle aged individual, the varicose veins are especially large, (2) when, in such an individual, a varicose ulcer is threatened or present, and (3) when incompetent communicating veins are proved to be a factor, especially in the presence of an ulcer. It may, in fact, be elected by patients in either of the first two of these three categories and is more or less mandatory in the last. The operation calls for eight to ten days in bed and perhaps two weeks in hospital. It requires a perfection of technique and gentle handling of tissues to secure ideal healing of a number of wounds, the least possible immobilization in bed, and unnoticeable scars.

The operation is performed under a spinal or gaseous anæsthesia, the foot of the operating table elevated perhaps six inches above the head. The same technique is employed for dividing the great saphenous vein at its junction with the femoral as is used in a high resection—an oblique incision,

parallel to the inguinal ligament, resection of all branches entering the upper stump, and ligation exactly upon the femoral vein. From its point of division to a point just below the knee, the great saphenous vein may be removed by any convenient method—the Mayo stripper, for instance. The operator may elect to tear the main saphenous stem from its branches and control hemorrhage by pressure, but he should prefer the more surgical method of cutting down upon the vein whenever a group of entering branches is demonstrable. In any case, he should keep in mind that the most important part of the operation is the abolition of back pressure from above by thorough eradication of the great saphenous vein from groin to knee. Here, with fresh instruments,\* the dissection of the lower leg is begun. As a rule, the course of the principal varicose channels in the calf should be studied and a liberal incision made which will permit their removal without lifting flaps for more than an inch to one and a half inches in a lateral direction. Unless for some special reason, the veins need seldom be removed for more than two thirds of the way from knee to ankle, if a wide dissection in the lower part of this area seems desirable, a transverse cut, making the whole incision take the form of an inverted T or broad Y, is wise. Since the arterial supply for the superficial tissues of the front and sides of the calf comes from longitudinal rows of small arteries emerging from the deep fascia at quite regular intervals, very wide flaps are likely to slough.

In making an incision for the removal of varicose veins, the dissection should at once be carried to the deep fascia (whether or not in so doing the veins are divided in several places). Then flaps of full thickness, including all fat and subcutaneous tissue, are turned up, and from the inner surface of the flaps the veins are removed. This step causes the least possible traumatism to the cutaneous edges and tissues in general, and favors healing without necrosis of the skin.

There is little more justification for using the same dissecting instruments for a succession of incisions in one patient than for using the same ones for patient after patient.

If a probable connection with the varicose lesser saphenous vein has been demonstrated—that is, a leaking communicating vein in the region of the popliteal space—a *transverse* incision should be made behind the knee. The lesser saphenous will usually be found without difficulty just beneath the thin deep fascia, passing upward in the middle of the space. No more of it than can easily be reached through the transverse incision need be removed.

*The Operative Removal of Varicose Veins in the Presence of Ulcer*—It is desirable that an ulcer should be clean if not healed before a radical operation is undertaken, since the lymphatics encountered in the dissection may, in the presence of dirty ulcers, become a source of infection.

The veins are removed as usual and the dissection is carried as near the ulcer as is considered safe. It is convenient to end the linear incision in a very broad inverted Y. If the ulcer is of moderate size and little indurated, this will be sufficient. But if the ulcer is old and much indurated, it may be excised at the same time with, or some days subsequent to, removal of the veins. In the former case, the excision of veins should first be finished and the wounds closed. Then the ulcer, with a margin of sound tissue *and the aponeurosis beneath* should be removed in one block, laying bare muscle and, if necessary, tendon sheath, periosteum, or the capsule of the ankle joint. The clean surface thus left can at once be covered with an Ollier-Thiersch graft which usually heals without difficulty. Such a radical procedure is seldom necessary. Most ulcers can be given a trial of excision of the veins alone. If that fails, it is time enough to remove them.

*After-Treatment*—When the patient is first allowed out of bed—some ten days after the operation—the leg is bandaged from toes to knee. Moderate exercise is encouraged but when the leg is not in use, it is elevated, not left dependent. A week of this routine should be taken to accustom the leg to new conditions, after which the bandage may gradually be left off.

Should an ulcer have been excised and a skin graft made, some weeks are required to accustom the graft to its depend

ent position. It tends at first to be very cyanotic and to break down at its edges. It must therefore be supported by a soft pad and a semi elastic cotton bandage.

**Rupture of Varicose Veins**—Occasionally and unexpectedly, a varicose vein ruptures externally, giving rise to a gush of dark blood. The vein which ruptures does not seem to be the great accumulated vessel projecting above the surface of the leg—such is more apt to undergo thrombosis—but a smaller and less conspicuous one, the wall of which, however, is covered only by very thin skin. Rupture always occurs below the knee. Naturally, for so long as the leg remains dependent, blood flows rapidly from the tiny opening and will in time, if unchecked, drain the entire vascular system. However, the leak is promptly stopped by elevating the leg above the body and making pressure upon the region of the hole. A firm bandage over a small gauze pad sufficiently compresses the varicose vessel so that little or no blood can reach the opening. Thus the individual, if given first aid treatment, can usually get about at once. It is then advisable, not as an emergency but within a week or two, to divide the vein above the rupture, whether or not the great saphenous is resected at the groin. Rupture is of course an indication for some sort of curative treatment of varix.

**Thrombophlebitis in Varicose Veins**—Those who have read, up to this point, the sections devoted to varix will have observed that almost no mention of thrombosis has been made. The apparent omission is due to the writer's conviction that all forms of thrombophlebitis should be considered together—that which occurs in varicose veins being the most common, least dangerous, and on the whole the most tractable form. The thrombophlebitis of varix will, therefore, be described in the following chapter. It is enough to say of it here that it is, next to ulcer, the most annoying and disabling complication of varicose veins. It occurs unpredictably in persons of all sorts, young, old, vigorous and feeble. Once it has attacked a varicose vein it is apt to return, like the tiger with a taste for human blood. Its treatment, because it is an everyday affair

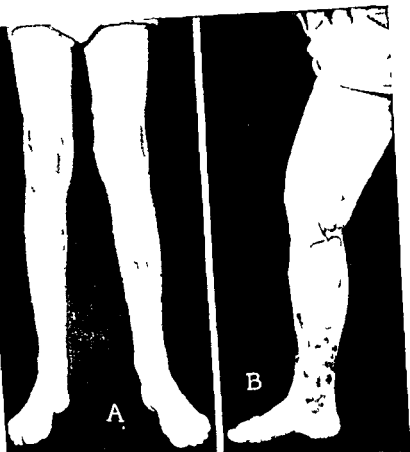


which almost never takes life, has long been unenterprising and dictated by outworn traditions Embolism is exceedingly rare but does occur

### POSTPHLEBITIC INDURATION AND ULCERATION

Little is to be found in textbooks or in the literature of ulcer upon the subject of the common disabling indurations and ulcers which follow phlegmasia alba dolens (femoro iliac thrombophlebitis) Leriche briefly describes and pictures them as results of a vasomotor disorder associated with this sort of thrombophlebitis In this country, Trout has noticed them and described an operative treatment The writer, in early papers (1916-17) primarily devoted to varicose veins and ulcer, described them as "the sort of ulceration which is associated with postphlebitic varix of the small vessel type" That is, he was unable to shake off the idea that some sort of varicosity was behind the ulcer, though he recognized the postphlebitic character of the disease Actually this was a useful conception, for it led to many efforts to cure the indurations and ulcers by excision of the dilated superficial veins and division of the communicating veins with which the lesions are so often associated But the dissections proved that though defects of venous drainage were a contributing factor, the lesions could and often did occur in their absence and were not in fact "varicose ulcers" This is the reason for the considerable space given here to these lesions Many practitioners, upon seeing something which appears to be a varicose ulcer, seek out any vein in the vicinity and inject into it a sclerosing solution By so doing they merely increase the local inflammation and aggravate the sore

Postphlebitic induration and ulcer follow only a deep thrombophlebitis No enlarged visible veins need be associated with them However, in a minor proportion of cases a number of superficial veins seem to become enlarged, as collaterals, during the obstruction of the deep vessels and afterwards become varicose Moreover, there may rarely be associated with them a straight, hard, shrunken saphenous vein which has apparently



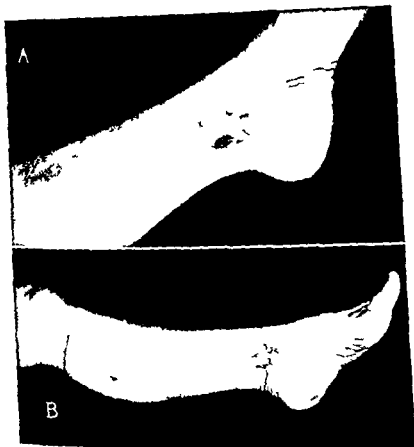
"VARICOSE VEINS" (1899) a man aged fifty. Bilateral varicose of inl vein type. The ulceration area is deep and most seeping. This might be an allergic reaction to fungous infection. B. M. C. 1910 a woman aged sixty. Varicose vein of large type and a red weeping area—allergetic! The ink mark below the knee indicates the proposed site for low division.

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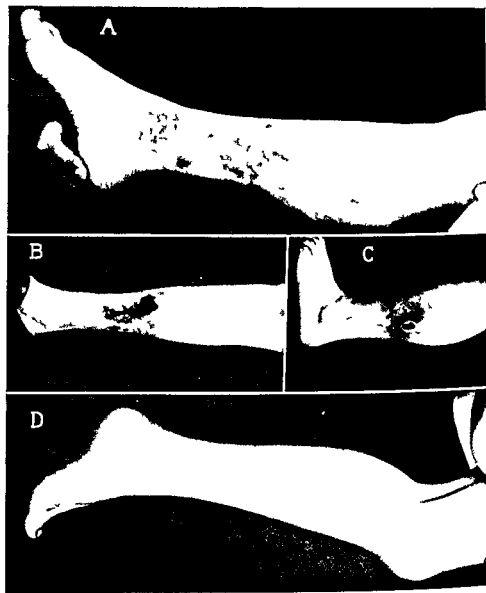
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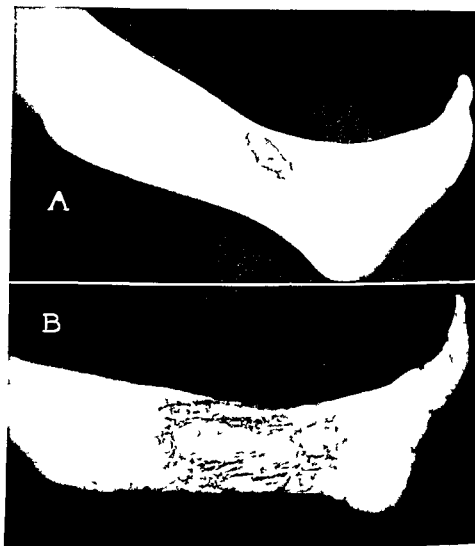


**POSTHEMORRHAGIC INDURATION AND ULCERATION** *A* D.G., 30728, a woman aged forty two. Secondary varix with unusual degree of sclerosis. Cure by excision of vein. *B* M.A.H., 3132, a woman, aged sixty five. Notice dilatation over sclerosed great saphenous vein secondarily involved in femoral deep thrombophlebitis. *C* M.C.O.D., 23076, a woman aged twenty three. Pigmentation and ulcer in an unusual situation. Successfully treated by excision and skin graft. *D* H.K., 59506, a woman, aged forty seven. Early stage of induration without ulceration. No veins visible. Elevation of flap showed no incompetent communicating veins. Treated by lumbar sympathectomy.

been thrombosed at the time of the femoral thrombophlebitis. And there are very likely to be associated with them incompetent communicating veins (whether or not dilated superficial veins are evident). Any or all of these may contribute to the persistence of postphlebitic induration and ulceration. However, as has already been explained, it is easy enough to find cases in which obstinate postphlebitic lesions are associated with no abnormal superficial or communicating veins whatever.

*Onset and Course* — Because the pathological features and appearance of the lesion itself are not entirely characteristic, its onset and course, which are peculiar, will first be described. The earliest signs of the disease appear at any moment from three months to twenty years following the femoro-iliac thrombophlebitis. The "milk leg", "phlebitis", or whatever it may have been called, will usually have been severe, but recovery will not necessarily have been, though actually it often is, succeeded by residual swelling. Without warning and usually with little discomfort, a patch of edema will be noticed upon the lower third of the leg, most often upon the inner face of the calf a few inches above the ankle, but occasionally upon the back or in the neighborhood of either malleolus. As a rule, the swelling is low, rounded, hot to the touch, very slightly reddened, and from two to four inches in diameter. One seldom sees the lesion at this early stage, since the patient does not consider it serious. Actually it is nothing but a local patch of edema, not a furuncle like affair, yet it is slightly hot to the touch as if it were the seat of a mild, non-suppurative infection. In some instances, the appearance of swelling is lacking and the skin is merely pigmented and faintly indurated. If pigmentation is the first sign, the color may deepen rapidly to a heavy brown. Rarely pigmented patches or indurated areas are multiple.

If seen very early, the local edema can usually be made to disappear upon elevation for a few days in bed. The writer has seen one or two cases aborted, as it were, by prompt elevation and bandaging. The following is an instance of this sort.



POSTTUBERCULIC INDURATION AND ULCERATION. I. S., 30133, a woman aged fifty-three. *A*—Small ulcer but larger area of pigmentation and induration. Note small dark area toward back of calf separate from the main lesion—each a local induration of fat. *B*—Two years after excision and Ollier Thiersch graft.

Except for its relation to phlegmasia alba dolens and its mode of onset, that is, through the establishment of local edema and induration in the absence of varicose veins, there is little to distinguish it from any other ulcerative process upon the legs. Pain is usually present, especially in the case of lesions upon the inner face of the calf, a little above the internal malleolus. Such ulcers have been called "irritable ulcers" and are often agonizingly painful.

Veins are rarely noticeable, at least at first, and, indeed, may never appear. However, as already explained, an occasional collateral enlargement may have ended in varicosity, so that a rather sclerosed type of varicose vein sometimes leads from the groin to the region of the induration and ulcer. In that case, varicosity clearly contributes to the development of the sore and gives an opportunity to test for the presence of incompetent communicating veins. These latter will often be found to be present, so that it has seemed to some as if they must be a basic feature of the disease. Their presence is discovered by using the second form of the Trendelenburg test, that is, the test of back flow when a constriction is applied to the thigh. On lowering the leg, after a turn of bandage has been tightened about the thigh, the veins of the calf will be felt to become tense in five to ten seconds, or even less, showing that venous blood is leaking from the deep veins out through the communicating vessels to the surface. But the foot does not become blue, for the deep veins themselves are not crippled.

Undoubtedly the leaking communicating veins are frequently a feature of postphlebitic ulcer, and one or more will be found (on exploration) beneath a great area of induration. However, the writer has excised many such areas in toto, including the aponeurosis beneath, without finding any perforating veins of an incompetent sort (one can test their competency at the operating table by observing whether or not they are grossly dilated and allow blood to flow toward the surface when cut). Indeed it has never seemed to him that the veins were greatly concerned with the pathology of the lesion.



A married woman, thirty three years of age, had suffered, seven years earlier, from a bilateral milk leg, the left by far the more serious. She had subsequently gone through a pelvic operation without a recurrence, but the left ankle had always swollen after a long day on her feet. For several days before she came under observation, she had noticed a localized swelling and hardness just above the external malleolus. This gave her some discomfort, especially at the end of the day. In other respects she was well.

Examination revealed an area of indurated edema several cm. in diameter, slightly reddened and barely elevated above the surrounding skin which, as compared with the other foot, was faintly pinkish blue in color.

A semi elastic cotton bandage was applied from toes to knee and the patient was advised to spend as much as possible of the next week in bed. This she did, with the result that the ankle took on a natural color and the edematous spot disappeared. She was instructed that the early lesion was a danger signal and that if at any time she expected to be much on her feet she should use her bandage. Two years later she reported that she had had no further trouble, barring some swelling of the ankle when her children kept her particularly busy. Whether early treatment should always produce this favorable result is unknown, for most patients present themselves only when the edema has become permanent induration or actual ulceration.

Once the lesion is established, it takes on a discolored appearance, usually a combination of pigmentation and redness which fades into the normal tissues about it. The skin is thickened, the subcutaneous tissues hardened. The hardness fades gradually, in some cases, into the normal soft quality of the subcutaneous fat. In others, it ends irregularly and abruptly so that its rather scalloped border, though invisible, can be palpated. At this stage, ulceration will usually have occurred at its center, a sore very much like the early varicose ulcer, having its same tendency to heal on elevation and protection, and the same tendency to widen and deepen with time.

calf On the other hand, the very early lesion, as in the case already quoted, may be made by elevation, rest and bandaging to disappear altogether, or, if more advanced, will by similar methods be held in check In some instances, enlarged veins, passing into the indurated area from above, can be removed with benefit, an operation which may include resection of the entire great saphenous system. At the same time any incompetent communicating veins encountered can be divided at the level of the aponeurosis Any such operation, it will be realized, will approach, if it does not actually enter, a field in which the tissues are indurated and perhaps actually infected. It is carried out exactly like the operation for varicose ulcer (*qv*) It will be best that any such operation should not be performed in the presence of an open ulcer—the tissues are already sufficiently liable to infection They must be handled with great gentleness, and asepsis must be perfect. Recently, Linton has shown how by long incisions from knee to ankle, carried through the aponeurosis, the area of induration can be elevated and the incompetent perforating veins divided from beneath.

Excision of the indurated, ulcerated area is a very satisfactory way of permanently curing the disease This does away at one moment with any venous stasis and the badly diseased tissues The operation should be reserved for cases with heavy and wide spread induration, should remove *all* scarred tissue, no matter how large the area, and should always include the muscular aponeurosis If this heavily scarred layer is not taken, a skin graft upon its surface will never permanently survive On the other hand, if the aponeurosis is removed, a successful graft can be placed upon the exposed periosteum, tendon sheath, muscle, or capsule of the ankle joint—the tissues beneath the aponeurosis not being involved The excision should be performed in a bloodless field (Esmarch bandage to thigh) After removal of the Esmarch bandage, the bleeding vessels should be tied with the finest silk. An Ollier Thiersch graft, immediately applied, will almost invariably “take” and survive, though its subsequent adjustment to a dependent posi-

The most noticeable *pathological feature* of postphlebitic ulceration is the thickening and hardening of the tissues, which is most marked upon the surface of the aponeurosis. Indeed, this layer is sometimes two to four mm in thickness, a dense leathery barrier through which the arterial supply fails to penetrate from beneath (the superficial tissues are supplied, as already explained, by arterial "trees" which pass from the great arteries out through the aponeurosis in a series of rows). If, then, one were to sketch what appear to be the steps which lead to the establishment of the advanced lesion, one would place them in the following order: localized edema (lymphatic or vasomotor?) associated with low grade infection, fibrosis, lowered resistance to infection, ulceration. After which the vicious circle of edema, fibrosis, diminished arterial supply, and infection continue and cause the lesion to extend. Sometimes, indeed often, after a great ulcer has been excised and skin-grafted, yet leaving a little induration at one edge, an acute process will recur at that point and soon involve new areas.

The diagnosis is suggested by the appearance of the lesion and the absence of a history of varicose veins. Indurations and ulcers unusually placed (from the standpoint of varicosity) or multiple are nearly certain to be postphlebitic. The accompanying page of illustrations will support the statement that the appearance of any one lesion should lead to an investigation of its background. See Plates VII, VIII and IX.)

*Treatment*—Since induration and ulceration may become established in youth, following the milk leg of childbirth and the thrombophlebitis of acute fevers, surgical operations and injuries, there is often granted an opportunity for cure at a time when the tissues are still capable of permanent healing. Individuals first seen when over fifty years of age, especially when the lesions themselves have been present for many years, are usually incurable. Indeed, there is nothing more resistant to treatment than a long standing postphlebitic ulcer which occupies the inner face of the leg from ankle half way to knee and encroaches on both the anterior and posterior faces of the

pathectomy, had better be treated by other methods. In the female, removal of the first lumbar ganglion is not only harmless but will raise the level of vasodilatation to the thigh

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tion is often time consuming. It is remarkable how soon the abrupt edge left by the excision of such an area is flattened and smoothed, showing that the edema and thickening about the ulcerated area has disappeared.

Other, less radical means of dealing with these postphlebotic lesions are (1) nerve division and (2) lumbar sympathectomy. The former is only available for painful, but not too deeply scarred processes upon the inner face of the calf, at or above the median malleolus. The latter is most likely to succeed in the case of lesions moderately indurated.

*Internal Saphenous Nerve Division* is especially useful in treating painful ulcers in the field of that nerve. The ulcer should be healed or at least very clean when the operation is performed. A transverse incision is made under procaine infiltration just below the crease on the inner face of the knee joint, that is, over the great saphenous vein which exactly overlies the nerve and is the guide to it. The nerve, in turn, lies upon the aponeurosis, exactly behind the vein, usually a single structure but sometimes in the form of two trunks (having split just above). Having divided the nerve, the operator will do well to bury its proximal stump beneath the aponeurosis. On the whole, the larger the nerve and the less it has broken up, the more clean cut and free from overlapping collateral sensory supply will be the saphenous field.

The effect of a saphenous block is to raise slightly the cutaneous temperature (partial sympathetic paralysis) in the saphenous field. The ulcer will become painless and will very often heal. (See Plate VIII.)

*Lumbar Sympathectomy* is only likely to succeed when in duration is of moderate grade, incompetent superficial and communicating veins are absent, and the vasodilatation of the sympathetic paralysis can be made to mount higher than the lesion. To cause vasoparalysis to reach up to the knee, excision of the first as well as the second and third lumbar ganglions will usually be required, an operation which may disturb the male sexual function. Therefore, in the male, indurations which extend high, though otherwise appropriate for sym-

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## CHAPTER VI

### THROMBOPHLEBITIS AND PULMONARY EMBOLISM

THE thrombophlebitis of surgical operation, accident and serious illness is recognized today as a problem of the first importance. This venous thrombosis is one that occurs, not in the auricles of the heart, the cerebral sinuses, or the portal system, but in the veins of the pelvis and lower limbs, a sort which attacks persons put to bed because of surgical operation, childbirth, injury, or disabling disease and which not only adds a new illness, but introduces the hazard due to the presence of a detachable thrombus. Beside this common variety, which might well be called the thrombophlebitis of hospitalization, there are other forms which occur in active life. Each has its anatomic background and each presents itself in a characteristic way.

The word, thrombophlebitis, is taken to mean thrombosis in a vein and does not necessarily imply inflammation, certainly not such inflammation as is caused by infection. "Phlebitis," so much used by the laity, is therefore a bad word, thrombosis being the basis of the process and phlebitis a reference to its scene. At its very start, thrombosis is not clotting, for clotting relates only to coagulation of the blood, a chemical biological process which follows fairly well defined rules, occurring when blood is exposed to tissue juices, after death or in association with the release of a thromboplastic substance from thrombocytes. Thrombosis can and usually does take place within normal intact blood vessels. The thrombocyte, or platelet, is the villain of the piece, attaching itself in places where its death will be most inconvenient. When a mass of these tiny discs becomes plastered upon the intima of a vein, they die, and a thrombus has begun to form.



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Blockage, new Principle in Treatment", *Arch Surg*, 18 2281, June,  
1929

unnecessary to follow what has become the classical form, and relate the almost innumerable causes of thrombosis assembled under Lubarsch's tripos, that is, disorders of the venous return, disorders of the blood, and disorders of the vein's wall. An account will be given rather of the factors which are recognized today as leading most directly to thrombosis and which are being attacked with some vigor in various parts of the world. These include (1) retardation of the venous flow, and with this certain basic anatomical relations, (2) dehydrating factors, (3) the local and general influence of trauma, which may be called the "X" factor, and (4) inflammatory changes about certain great arteries and veins.

In the discussion of thrombophlebitic influences which follows, reference will necessarily be made to several varieties of thrombophlebitis. These will subsequently be fully described. It is sufficient here to give a brief characterization of each.

*Femoro iliac Thrombophlebitis Phlegmasia Alba Dolens Milk Leg*—This is the common "phlebitis" which is responsible for the great, white, swollen leg—usually an outspoken disease, sometimes painful, especially at its onset. Its scene is the principal vein draining the leg in the region of the groin. Only a very small proportion of femoro iliac thromboses lead to pulmonary infarction or serious embolism. Yet the disease is so common that the number of resulting embolisms must be considerable.

*Thrombophlebitis in the Deep Veins of the Lower Leg*—This seldom recognized disease affects the great plexuses of veins in and among the muscles of the calf\*—a system required for emergency use and therefore capable of nourishing, with little external sign of congestion, a silent, treacherous process—a frequent cause of embolism.

*Thrombophlebitis in Nonvaricose Superficial Veins*, a freakish process, due to trivial causes, often local but an occasional cause of a propagating thrombus and at least minor pulmonary embolism.

\* A good idea of the capacity of these veins can be had by consulting Figure 13 (Chapter V) and Figure C of this chapter.

The thrombus grows into the venous stream as a sponge like mass of dead platelets, which excites coagulation, so that leucocytes, red cells and fibrin are soon entangled in its tough meshes. At first the process does not close the vein but when the body of the thrombus has been built out from the adherent head, the vessel is soon filled with a mixed, dark red solid mass. As the thrombus extends up and down the vein, its youngest portion, or tail, is seen to be soft and clot like having no longer a supporting framework of platelets. Indeed, the tail of a thrombus is almost pure coagulum, red and flimsy, easily broken up and carried away. A thrombus tends to grow until it meets a vigorous stream, and thus its proximal end is likely to heal at the point where a branch carrying a strong current enters the thrombosed vessel. Its extension distally is uncertain, since it is not easy to say where a good current will be able to leave the main vessel against the set of the valves in the entering branches. However, there are many curious bypaths in the venous system, and doubtless the establishment of collateral channels is less difficult than one would suppose. In a complete backwater, the intima being intact, a thrombus does not form. It is the slow and feeble current which offers it encouragement. Into such a sluggish stream, proximal to the main thrombus, the flimsy tail grows on, often projecting from a smaller vein into a greater or along a greater in the slow, entering current of a smaller—a propagating clot.

Thus propagating clot, soft and fragile, waving free in a large vein, such as the femoral or external iliac, is the source of pulmonary embolism, for the embolus, once broken off, meets with no obstacle from its point of detachment in its course through the great iliac vessel, the vena cava, the right side of the heart, and so into the pulmonary artery.

It is easy enough to grasp the nature of the thrombus, its solid head, occluding mixed body and insecure tail. But why do thrombocytes lay themselves down to die in particular parts of the venous tree? And why, under circumstances equally favorable to thrombosis, does a thrombus form in one individual and not in fifty more? In presenting this problem, it seems

confused, encouraging the settling and adherence of thromboytes. Another area, more commonly the scene of thrombophlebitis than is generally supposed, is the popliteal region and the upper part of the calf. For several plexuses draining the great flat, flexor muscles come together here.

Most, if not all of the causes of a slow venous return disappear when the legs are elevated, especially if the thighs are not flexed but in line with the body. For this reason, an obvious first step in thrombus prevention is elevation of the foot of the patient's bed. Already some observations have been made upon the favorable effect of such treatment, especially in gynecologic surgery, and since many surgeons are attempting by this method to discourage postoperative thrombosis, positive information of its value will soon doubtless be available. There is also evidence that a thrombosis once started ceases to progress toward the heart when once it meets a vigorous current. For example, in the varicose saphenous vein it frequently ascends to the junction of that vein with the femoral, which, however, it is almost never able to enter. The writer has observed, moreover, on several occasions, the effect of elevation upon superficial thrombosis in a nonvaricose vein. In an individual whose thrombosing process had just run over both legs while he was allowed to recline in bed, a new thrombosis was halted and disappeared within a week when the foot of the bed was elevated six inches.

By contrast, as already explained, the dangerous propagating thrombus, the source of fatal pulmonary embolism, is fostered by a slow current. That is to say, the friable, clot-like tail of the thrombus grows out into a feeble stream entering from a proximal branch or, if thrombosis has begun in a branch, the tail grows into and waves in, without occluding, the sluggish current of the principal vein. Until the propagating thrombus has been studied further, the conditions under which it forms and grows can only be surmised, yet there is evidence, especially in instances of thrombosis in the deep veins of the calf, bearing upon the matter, as will appear in the following accounts.

*Thrombophlebitis in Varicose Veins*, a common disease, in capacitating but not dangerous. The thrombosis is usually solid and strictly confined to the varicose vein—rarely a source of embolism.

Thrombosis also occurs in the venous plexuses of the internal genitals of both sexes, the prostate and the uterus, but the behavior of the process is little understood and its clinical nature is unknown. It is probably a source of serious pulmonary embolism.

*Retardation of the Venous Return*, that is, a slow but not a dead current, is essential to thrombosis. In surgery, the retardation is usually due to mechanical causes, in medicine, to debilitating disease or enfeeblement of the heart. But in any case, confinement to bed is of first importance. Certain anatomical relations are of hardly less moment. Blood is pushed out of the lower limbs by muscular action. Therefore, a patient reclining or sitting up in bed causes the return flow from the relaxed legs to be decidedly delayed. Add to this effect the increased abdominal tension of intestinal distension, post-operative or otherwise, of tight abdominal dressings, and of excessive adiposity. Or add to it the pelvic venous congestion of pregnancy and the puerperium. Such influences aggravate the inherent difficulty of emptying the veins of the legs and pelvis unless the lower part of the body is raised above the upper.

Certain anatomical features not only retard the venous blood flow but introduce those eddies and cross currents which, according to many, favor a deposit of blood platelets in certain localities. The relation of the iliac veins to the great arteries of the pelvis is sufficiently familiar. The left common iliac vein is crossed at almost a right angle by the right common iliac artery. There is thus a hypothetical slowing of the venous current in the left common iliac vein. The slowing is perhaps most likely to produce its effect where the vein passes behind the hypogastric artery. In the region of the groin, many branches enter the femoral and external iliac veins. Such valves as may be present are large. Here, then, the current is

which are not obstructed by the initial thrombosis. It is very probable, though by no means certain, that the same result is to be expected in a majority of similar cases.

To sum up the disorders of the venous return the difficulty of forwarding blood from the legs is increased by a reclining position in bed, especially if the legs are flaccid, it is aggravated by increased abdominal tension and pelvic congestion, and because of confused currents in the upper calf and at the groin, a slow stream is especially likely to lead to thrombosis at these points, finally, a slow current, once thrombosis has taken place, favors the formation of a dangerous propagating clot, just as a brisk current discourages such a process.

Dehydration has long been recognized as leading to thrombosis. Individuals who have become anemic because of bleeding uterine fibroids, those who have become depleted by vomiting, by sweating and failure of fluid intake in connection with an abdominal operation, perhaps those also who have suffered serious malnutrition from any cause, are more liable than others to thrombophlebitis. Dehydration must, however, be counted among the influences which occasion clotting rather than thrombosis, in the strict sense of the words, influences which Bancroft, of New York, has described so well and for which he has established a "clotting index." On the whole, dehydration and depletion of the blood in general are the least obscure and most remediable of all the various causes of thrombosis under discussion. Means of prevention, of course, can readily be practised. The free administration of parenteral fluids, if the intestinal tract is not sufficiently available, and, when necessary, transfusion of blood, offer the obvious remedy. Further discussion of thrombophilic influences in the blood and body fluids will be found in the following section.

Trauma, the "X" factor, which seems to be the immediate excitant of thrombosis, is not only among the most active but certainly the most elusive of all thrombophilic influences. Its importance today is attested by the fact that such measures as are directed against thrombophlebitis are being used immediately after the operation, childbirth, or accident. Though

A man, fifty years of age, suffered a fracture of the fifth metatarsal bone in a minor accident. A plaster cast was applied for a week. For the next four months swelling of the ankle and moderate discomfort in the calf occurred repeatedly upon use of the leg and rapidly disappeared upon rest in bed. At the end of this period he died of pulmonary embolism. The twelve to fifteen inch (thirty to thirty five cm) embolus was found to have been detached from the point at which a large thrombosed vein entered the popliteal, as shown in Figure 22C. The femoral vein had never been occluded. Here was a fatal, easily detached, propagating clot, encouraged to form by partial occlusion of, and retardation of the current in, the venous tree and never subjected to continuous elevation. By contrast, prompt elevation following the establishment of thrombosis in these same vessels seems to have a favorable effect and is likely to cause the thrombosing process to recede and heal. The following is a case in point.

A vigorous athletic man, twenty seven years of age, was first seen when, some four days after jumping a brook, one leg had become lame, the ankle and calf considerably swollen but without any ecchymosis. He displayed the sign which the writer believes to be characteristic of this disease, that is, a painful soreness noticed high up in the back of the calf upon forced dorsiflexion of the foot, by which the tendo Achillis is put on the stretch. The foot of his bed was elevated six inches, the leg placed on a soft pillow but not immobilized. In a week, the swelling had disappeared. In ten days, all soreness on passive dorsiflexion of the foot had gone, and in three weeks the patient was going about as usual. There has been no recurrence.

This, like the preceding case, is judged to represent thrombosis in one or more of the great intermuscular plexuses of the lower leg. But here, instead of resulting in the formation of a propagating clot and death from pulmonary embolism, the thrombosing process healed to be organized and absorbed. This favorable outcome is laid to elevation and the establishment of a brisk venous current in the many veins of the region.

and fibula. The suggestion has been offered, without proof, of course, that some product—allergic or otherwise—of damage to, or rapid atrophy of, muscle, consequent upon the injury, is the active, exciting thrombosing influence.

There is then, perhaps, a state of the blood, related to the general and local effect of trauma, which may be expected to act rather rapidly. The conclusion is difficult to escape that when other influences are favorable, the injury determines the decisive action of the thrombocyte. Best and his associates in Toronto have attacked thrombocytic adherence directly, making use of a perfectly pure heparin, which they have developed themselves. By intravenous therapy, based upon animal experimentation, they have a reasonable hope of preventing the adhesion of the thrombocyte to a venous surface. Sufficient heparin to raise the clotting time to about fifteen minutes has been given continuously for several days, beginning an hour or so after operation, in a long series of major procedures. Apparently, postoperative hemorrhage is not a danger and thrombosis is abolished. Similar observations, which have not as yet been sufficiently prolonged to lead to an authoritative conclusion, are being made by Crafoord in Stockholm. One may say, then, that hypothetical changes in the blood, related to trauma, are important enough to suggest the need of using measures directed against thrombosis immediately after the operation or other injury. This, of course, is not at all new. Previous attempts, such as the administration of desiccated thyroid gland, exercises in bed, and, more recently in Europe, "sympatol" and other substances have been used in a similar way.

*Perivenous Inflammation*—Changes in the vein's wall, brought forward as one of the causes of thrombophlebitis, have been thought to represent degenerative and inflammatory reactions. In the presence of acute fevers, this seems intelligible enough, and indeed acute rheumatic changes, actually leading to thrombosis, are recognizable. However, evidence of such lesions in the vein's wall as a consistent cause of the familiar, and especially the postoperative, forms of throm



the gross evidence of thrombosis, that is, edema in the form of phlegmasia alba dolens, may not appear until two to three weeks or even longer after operation, there is good reason to believe that the actual onset of thrombosis and of the conditions which lead to it occur much earlier. Moreover, if the propagating thrombus must, as Aschoff states, attain a length of some twelve inches (thirty cm) or more to become a fatal pulmonary embolus, it clearly requires time\* for its formation, presupposing an onset of thrombosis very early indeed. Thus, although the proper combination of factors can surely occasion thrombosis at any time, the early hours and days are especially liable to it.

The thrombophilic influence of trauma is, apparently, both general and local. There is no intelligible reason why an operation upon the gall bladder should cause a thrombosis in the left upper femoral and external iliac vein (its favorite seat). Yet unless the writer has been completely deceived, he has seen a locally reactionless thrombosis start in such a patient on the night of an utterly uncomplicated cholecystectomy and lead to a fatal embolism three days later (as proved by autopsy). Here the very fact of operation, barring, perhaps, increased abdominal tension, seems to have been the only exciting factor. But it is well known, also, that thrombophlebitis following fractures of the bones of the thigh and leg almost always occurs on the injured, not the sound, side. This is brought out in Vance's account of the fatal embolisms of accident cases observed over a period of ten years by the Chief Medical Examiner's Office in New York City. He reports that, in the large majority of instances, the causal thrombosis was found on the side of injury, however slight that might be. But one does not need to assume that actual damage to the vein itself is responsible, since fractures of the femoral neck are nearly as apt to lead to thrombosis as are those of the tibia.

\* Fatal embolisms have been known to occur on the evening of the day of operation. They are by no means rare on the third or fourth day. Therefore a long propagating thrombus must be able under the right conditions to form with great rapidity.

and fibula. The suggestion has been offered, without proof, of course, that some product—allergic or otherwise—of damage to, or rapid atrophy of, muscle, consequent upon the injury, is the active, exciting thrombosing influence.

There is then, perhaps, a state of the blood, related to the general and local effect of trauma, which may be expected to act rather rapidly. The conclusion is difficult to escape that when other influences are favorable, the injury determines the decisive action of the thrombocyte. Best and his associates in Toronto have attacked thrombocytic adherence directly, making use of a perfectly pure heparin, which they have developed themselves. By intravenous therapy, based upon animal experimentation, they have a reasonable hope of preventing the adhesion of the thrombocyte to a venous surface. Sufficient heparin to raise the clotting time to about fifteen minutes has been given continuously for several days, beginning an hour or so after operation, in a long series of major procedures. Apparently, postoperative hemorrhage is not a danger and thrombosis is abolished. Similar observations, which have not as yet been sufficiently prolonged to lead to an authoritative conclusion, are being made by Crafoord in Stockholm. One may say, then, that hypothetical changes in the blood, related to trauma, are important enough to suggest the need of using measures directed against thrombosis immediately after the operation or other injury. This, of course, is not at all new. Previous attempts, such as the administration of desiccated thyroid gland, exercises in bed, and, more recently in Europe, "sympatol" and other substances have been used in a similar way.

*Peri-venous Inflammation*—Changes in the vein's wall, brought forward as one of the causes of thrombophlebitis, have been thought to represent degenerative and inflammatory reactions. In the presence of acute fevers, this seems intelligible enough, and indeed acute rheumatic changes, actually leading to thrombosis, are recognizable. However, evidence of such lesions in the vein's wall as a consistent cause of the familiar, and especially the postoperative, forms of throm-

bophlebitis is not forthcoming. Moreover, there seems to be no difference, either clinically or pathologically, between the thrombophlebitis complicating acute fevers and that associated with traumatic, postoperative and puerperal states. Evidently infection and fever, as related to the operation or injury itself, are not important etiological factors in thrombophlebitis. Nevertheless there is a relation between *perivascular* inflammation and thrombosis, a relation unfortunately not often discernible but of such a nature as to make it clear that the inflammation, when present, is a cause of thrombosis and that thrombosis does not occasion the perivascular inflammation.

Perivenous inflammation has been discussed by Leriche, not so much as a cause of thrombosis as of the pains, edemas, and late complications attendant upon femoro iliac thrombophlebitis. Just as involvement of vasomotor nerve fibers by inflammation of the wall of a large artery leads, he believes, to disorders of the peripheral arterial circulation, so inflammation about a great vein, by involvement of perivenous nerves, causes peripheral pain, edema, and eventually, as a late complication in some cases, ulceration. Leriche comes to this conclusion partly upon experimental grounds but chiefly because of noticing the favorable effect of resecting, or perhaps merely freeing, adherent, chronically thickened, occluded iliac veins upon peripheral pain and edema. The writer is able to present a somewhat different sort of evidence. Being interested in the relation of the lymphatics to the edema of phlegmasia alba dolens, he has, in several instances of this disease, explored the pelvis in order to examine the lymphatic bearing tissues surrounding the great iliac vessels. In one instance of a left sided phlegmasia alba dolens, which had already persisted unchanged for about ten days, he found, upon dividing the peritoneum over the iliac vessels, an intense, nonsuppurative, vascular exudate surrounding both artery and vein. The artery was so engulfed in inflammatory tissue that it could not be seen, but it was evidently in a state of spasm. The vein was only found by cutting into it. Strangely enough, its intima was, by contrast with the adventitia, entirely normal and it

was filled with a solid, dark, mixed thrombus. It was clear that a primary perivascular inflammation had led at one moment to venous thrombosis and arterial spasm, and inconceivable that the reactionless thrombus could be the cause of the active, external, inflammatory exudate. The perivascular reaction surrounded the common as well as the left external iliac vessels and was found to extend down Hunter's canal. In the lower third of the thigh it was much less active, and here the femoral vein was found to contain liquid blood. In another case of longer duration a similar reaction was seen during the stage of repair, the exudate having undergone such fibrosis as to unite the artery and vein firmly to each other and to the surrounding sheath. Yet it cannot be shown that the perivascular reaction is always present, for in a third exploration by the writer, there was no trace of it, though a solid thrombus occupied the left external iliac vein up to the crossing of the hypogastric artery. Obviously, if such a reaction is the rule, it will be necessary to offer an explanation of its origin. One may, therefore, go back to the lymphatics, the primary object of the writer's explorations. Any infection carried by the lymph stream from the legs, the genitals, or the anal region must pass to the lymphatic vessels and nodes about the great iliac blood vessels. And there are many observed instances of lymphangitis associated with the onset of phlegmasia alba dolens. One such case is cited.

A youngish man, suffering from pneumonia of no great severity, exhibited a lymphangitis and femoral adenitis following hypodermoclysis of saline solution in the right thigh. The severe pain in the right leg, which then suddenly set in, was associated with disappearance of the pulses in the right foot and enfeeblement of the femoral pulse. Only after these signs of arterial spasm had occurred did the typical swelling of a femoro iliac thrombophlebitis appear. Unfortunately this patient could not be followed long enough to determine the further course of the phlegmasia alba dolens.

Here the matter must be left. The more interested one becomes in perivascular inflammation as a cause of femoro iliac

thrombophlebitis the more important it seems, but the various other causes of thrombosis are clearly so significant that one fears to become too much attached to one—and that little studied—hypothesis. However, a quotation from Cruveilhier, upon the subject of iliac thrombophlebitis, is perhaps appropriate.

“D’une autre part, j’ai vainement cherché dans la membrane interne des veines des traces d’inflammation point d’injection vasculaire, point d’épaississement notable. On ne trouvait d’indices de travail fluxionnaire que dans le tissu cellulaire *exterieur* aux veines, lequel tissu était plus cohérent que de coutume et injecté de capillaires veineux qui enlaçaient la veine comme dans un réseau et pénétraient dans son épaisseur. Dans aucun cas de phlébite, je n’ai trouvé la membrane interne des veines injectée.” (Itches, winter’s)

**Pulmonary Embolism.**—Before going on to the specific varieties of thrombophlebitis, it will be appropriate to discuss the nature of pulmonary embolism. The *Propagating Thrombus* has already been pictured as the usual source of embolism. There is ample reason to believe that, if it is to form, there must be available, proximal to the primary site of the thrombus, a sufficient length of large vein. That is, if the solid part of the thrombus ends proximally where the external iliac vein joins the hypogastric, the propagating thrombus must float in the common iliac and far up the vena cava. Aschoff believes that most long, fatal emboli come from the femoral, on the assumption the more reasonable because of the fact that outspoken phlegmasia alba dolens, which represents a thrombosis extending proximally through or above the external iliac vein, is seldom a source of fatal embolism. Certainly, many long emboli are broken off in the popliteal vein, having grown into the femoral as propagating thrombi, whence the importance of thrombosis in the deep veins of the calf.

In regard to the pelvic plexuses as sources of emboli, little is actually known. Thrombosis has been shown to extend, as an occluding process, from the uterine veins into the hypogastric and common iliac. But the unanswered question is: Does

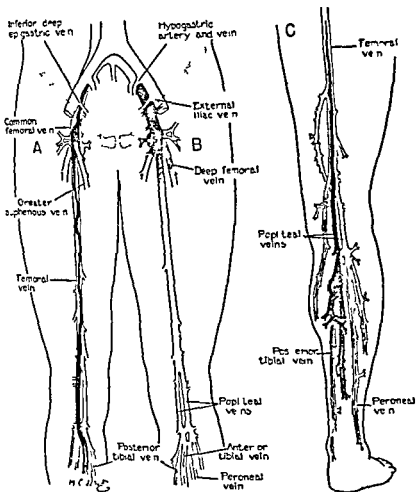


FIGURE 29 THROMBOPHLEBITIS IN THE FEMORO-ILIAC REGION AND DEEP VEINS OF THE LOWER LEG—Semidiagrammatic A A non-obstructing thrombophlebitis such as may be encountered in exploration of the common femoral. It causes little or no swelling and seriously threatens pulmonary embolism. In such a case, the common femoral must be divided and the clot gently sucked out. B Phlegmasia alba dolens, obstructing, and a cause of marked swelling. Entering branches more or less involved. Peripheral extent vague. Seldom causes embolism. C Thrombosis of the Deep Veins of the Lower Leg A reconstruction of an actual case in which fatal pulmonary embolism occurred. The long propagating thrombus broke off in the lower femoral. An early division of the superficial femoral vein would have prevented this accident.

a floating, propagating thrombus originate in the uterine or prostatic veins and grow out into the hypogastric and common iliac? If so, it may well break off, leaving little trace behind. Apparently there is no evidence that such occurs, though when no other source of embolism is discovered, the great pelvic plexuses must be under suspicion. Indeed, it is then hard to see from where else the embolus can have come.

*Embolism from unnoticed Thrombosis*—There is good reason to believe that outspoken processes such as phlegmasia alba dolens or thrombosis in varicose veins are less apt to cause embolism than the quieter ones. In the case of phlegmasia alba dolens, inflammatory changes, such as have already been described, tend to fix the thrombus, and the collateral current called forth by the obstruction of a considerable length of the main venous trunk would seem to offer little opportunity for the formation of the long, fragile, clot like, detachable tail. Again, in the case of varicose veins, the sclerosed state of the vein, the overstretching of its coats by rapid dilatation, perhaps the presence of bacteria in its wall, combine to make a thrombus adhere solidly. Common as it is for thrombosis, beginning in a sacculaton of the upper calf or thigh, to reach the saphenous opening, the extension of a soft propagating clot from the varicose saphenous vein into the femoral is almost unknown. Conversely, among Vance's ninety fatal accident cases of pulmonary embolism, already mentioned, there was rarely any external evidence that thrombosis had occurred. Another pertinent observation relates to the trivial injury as a cause of a quiet thrombosis and a fatal embolism. Vance noticed that among eleven such cases, in six, the thrombosis was on the side of the injury, while in the other five, no source of embolism could be discovered—facts which speak for the ease with which thrombosis can be established by trauma and for the increased frequency of embolism when thrombosis is not extensive enough to have caused external signs, or, for that matter, to have left internal traces.

It seems that not only may a short though dangerous thrombosis remain utterly silent, but even an extensive thrombosis

may be remarkably quiet. The reason for this is not entirely clear but a very gradual spread of the thrombosis is the probable explanation, for the collateral circulation can then keep pace with the obstruction. The writer has seen, in a vigorous elderly man, reclining in bed following prostatectomy, repeated pulmonary infarctions when only a very slight cyanosis of one foot was all that indicated the presence of thrombophlebitis. But when the attempt was made to divide the femoral vein at the groin, thrombosis, incompletely obstructing the vessel, was found. A fatal pulmonary embolism followed, and autopsy revealed a bilateral thrombophlebitis which extended proximally, on one side, as high as the common iliac vein. It is certain that incompleteness of occlusion by the thrombus is an important factor in the formation of a propagating clot. The writer has several times met with it in his attempts to divide a vein proximal to the supposed source of embolism. A slow blood flow past a soft thrombus should certainly offer an ideal soil for the creation of loose detachable fragments.

If the observations of Leriche, Cruveilhier and the writer, in respect to outspoken femoro iliac thrombophlebitis, are taken seriously, and if the frequency of embolism from quiet or unnoticed thrombosis is at all what has just been suggested, it is fair to suppose that with outspoken phlegmasia alba dolens, an insecure, embolism threatening thrombus is very unlikely to form, while with quieter, incompletely obstructing thromboses it is always a threat. The explanation is offered that the less the vein's wall is inflamed or injured previous to the onset of, and during the course of, thrombosis, the greater is the probability that an insecure propagating thrombus will be established and that a fatal pulmonary embolus will occur.

#### VARIETIES OF THROMBOPHLEBITIS

**Femoro iliac Thrombophlebitis Phlegmasia Alba Dolens Milk Leg**—Though instances of femoro iliac thrombosis have been observed in persons leading an active life (Barker) the



disease usually attacks those who, for one reason or another, are confined to bed. The veins involved are the upper femoral, the external, and perhaps common iliac. Apparently the confused currents due to entering branches and large valves favor thrombosis in this region. Aschoff remarks that when both sides are affected, the process is likely to extend, on the left, through the common iliac vein, up to the point where the vein passes under the common iliac artery, but on the right, mounts only to the region of the inguinal ligament. The anatomical background for this distinction has already been discussed.

Phlegmasia alba dolens is particularly apt to follow operation or injury in persons in or beyond middle life. But it occurs so often in young women after childbirth and often enough in young adults of either sex after acute fevers or such a simple operation as appendicectomy, as to make any categorical statement about a particular age incidence absurd. It would be better to say that a femoro-iliac thrombosis comes to mind when any sort of operation is proposed for an individual of fifty-five years or over. After an operation upon the prostate or hysterectomy for fibroids, it is perhaps twice as frequent as after upper abdominal procedures. Surgeons are apt to forget how very often a phlegmasia alba dolens is associated with debilitating diseases—not only the serious, acute fevers like pneumonia and typhoid but the circulatory failures and advanced organic diseases of any sort. Fractures of the lower limbs occasionally bring it on, the injury, plus immobilization of the leg, plus the reclining position so often used combining to offer an ideal background for the disease. Reasons have already been given for believing that the initial thrombosis usually starts very promptly after favorable conditions for its establishment have occurred. How soon it shows itself thereafter seems to depend upon developments little understood.

When a whole lower limb is swollen, it is certain that a femoro iliac thrombophlebitis is present, but absence of edema is no proof that the disease does not exist. The amount of edema seems to depend upon the extent of the process, the im

permeability of the plug, the efficiency of the collateral circulation, and the degree to which the deep lymphatics are obstructed. Naturally, a very extensive thrombosis makes the prompt establishment of a collateral circulation a difficult matter, whereas a local one is easily circumvented. An insidious onset and development is consistent with an absence of edema. By contrast, a very acute, active process will cause the limb to swell so tensely, within a period of seventy-two hours, that it will not pit on pressure and is utterly unwieldy. A leg of this sort is not cyanotic and though a sufficiently widespread thrombosis is perfectly capable, by itself, of causing an advanced edema, one cannot help thinking that lymphatic obstruction is very much concerned with the swelling. Paine looks upon an almost pure lymphedema as being exceedingly common. The writer, though understanding the perivascular inflammation which Cruveilhier, Leriche and he have demonstrated in their several ways, to be a probable cause of lymph edema, is aware that a very full swelling of a limb can be present without any perivascular involvement of the lymphatics whatever. This de Takats has experimentally proved.

The onset of phlegmasia alba dolens is more often than not ushered in by pain, sometimes of such severity as to suggest acute arterial ischemia. The pain is referred to the groin, the inner face of the thigh, even to the back of the knee or calf. With this there is apt to be some degree of soreness over the upper femoral region and sometimes above the inguinal ligament. But the swelling may appear insidiously, without pain or at the most with a feeling of numbness or heaviness. As a rule, the pain is preceded for twenty-four hours by a moderate elevation of pulse and a slight rise of temperature. It is not followed by swelling for perhaps another day. Then edema mounts rapidly from the ankle to the groin.

The behavior of the local pulse is of great interest. Allusion has been made earlier to a weakening or disappearance of pulsation in the peripheral vessels and even in the femoral. Evidence of arterial spasm is appearing more and more often in the literature. French observers, in particular, stimulated

perhaps by the writings of Leriche, have described arterio spasm so severe as to cause gangrene of the leg. Apparently it is not necessary that a perivascular inflammation should be the cause of this spasm. The mere presence of a thrombus in the upper femoral and iliac vein is sufficient. A very striking account is that of Gregoire.

A woman, forty-nine years of age, had been operated upon for pyosalpinx. Three days after operation, swelling and heaviness attacked the left leg but so mildly that the patient did not at first complain. On the following morning, however, the leg turned bluish, cold, and numb. In an hour, the color had reached the knee and soon the thigh as well was cold, cyanotic and rather marbled, a picture of arterial embolism. At the same time, the face became pale, the pulse rapid and the temperature elevated ( $38.5^{\circ}\text{C}$ ). There was no edema and strangely enough, no pain. Cutaneous sensibility was lessened. No pulsation could be detected in the femoral distal to a point three cm. below the inguinal ligament—the usual findings when the femoral is obstructed at its bifurcation by an embolus. Upon exploration, the femoral artery was found to be in a state of violent spasm for a distance of only two cm. and contracted to the size of the radial. This contraction lay beside the lower end of an enormous, bluish black stretch of femoral vein. The thrombosis ended cleanly three cm. below the inguinal ligament and disappeared beneath the ligament, above which it was not followed. Distal to the thrombus, the vein was small and pale. When punctured by a needle, the contracted artery bled in spurts. Evidently it transmitted a little blood and an injection of novocaine into its outer coats caused it to expand to its natural diameter, bringing back the peripheral pulse. However, the vessel soon contracted to its previous size, nor could it again be made to dilate in spite of a periarterial and perivenous sympathectomy over a distance of ten cm. (four inches). Perhaps spasm had now set in higher up. In any case, during the following days the leg became gangrenous and eventually was amputated above the knee.

It should then be recognized that venous thromboses may be associated with, if not actually a reflex cause of, arterial spasm serious enough in some cases to induce gangrene of the limb, and that pain, which usually marks the onset of arterial ischemia, may occasionally be replaced by mere coldness and numbness.

Minor degrees of this reflex sympathetic disorder are not so uncommon. Leriche has made the very interesting observation that it can be broken up by injections of novocaine into the paravertebral lumbar sympathetic. He states that by daily injections discomfort is diminished and that the edema disappears far more rapidly than the ordinary expectation. Whether such treatment should be made a routine in phlegmasia alba dolens or should only be used when evidence of reflex arterial spasm is observed is not yet clear.

The course of a femoro iliac thrombophlebitis is extraordinarily variable. A mild form causes only a moderate swelling which disappears after ten days and leads to almost no residual edema when the patient first gets about. A severe form, associated with fever and both local and general discomfort, results in a huge, hard leg which changes little over many weeks or even months. When at last the swelling goes down, the patient is sometimes left with a limb larger than its mate and subject throughout life to some degree of edema, especially of the ankle and lower leg. Once a femoro iliac thrombosis is established, some individuals seem to become increasingly thrombophilic. The opposite limb is involved far more often than is generally supposed, but perhaps with so little swelling that the second process is overlooked. Occasionally a thrombophlebitis passes back and forth from one leg to another, recurring later in the one first attacked.

The appearance of the leg is usually one of white swelling. There may be a faintly cyanotic, pinkish color as if there were present a generalized peripheral vasoconstriction. Veins are rarely visible but as the swelling goes down, large, seemingly collateral vessels sometimes appear in the upper thigh, the pubic region and the lower abdomen, remaining throughout

life Sometimes the great saphenous vein can vaguely be felt as a tender thrombosed cord which later becomes varicose Far more often, tenderness over the upper femoral canal gives rise to the suspicion of a superficial thrombophlebitis which, in fact, does not exist The leg is not noticeably hot or cool Only very occasionally are any lymphatic streaks to be seen

The aftermath of a phlegmasia alba dolens is usually far less disabling than might be supposed It is inconceivable, of course, that any valves involved in thrombosis can again function normally, yet very few feet are left cyanotic after the upright position is resumed The ankle may be puffy but the toes are not blue Only occasionally, large veins are left which in time become varicose The probability is that the worst of the thrombosis takes place in the external iliac, where only one valve is occasionally present, and in the upper femoral where after all only a few are lost Collateral vessels help out and the deep veins of the *lower* leg are seldom involved The real difficulty is with the superficial tissues Whether or not these are left edematous, there may develop later areas of edema, of pigmentation, of redness, of induration, and finally of ulceration, the "postphlebitic induration and ulceration" the nature of which is so obscure and the exact cause of which is so hard to understand Very rarely a painful hypersensitivity, associated, perhaps, with signs of vasospasm is left, a state reminiscent of causalgia (*q v*)

*Treatment, Preventive* —Certain influences favoring thrombosis are unavoidable in particular, the anatomic and physiologic peculiarities of the venous return from the legs, the exciting factor of operation, injury, childbirth, or debilitating disease, and of course life in bed To these are added the more or less avoidable influences of dependency and immobilization of the lower limbs, increased abdominal tension and dehydration The first set can be minimized, the second, in most cases, eliminated Elevation of the legs opposes the anatomic and physiologic difficulty with the venous return The legs and indeed the body in general can be exercised, preventing relaxation and atrophy Increased intra abdominal tension can

be forestalled by perfect closure of wounds, loose dressings and the skillful anticipation of intestinal distension. The difficulty with all such measures is that in nineteen cases out of twenty they are not required—one must go gunning for the twentieth case! One must never, however, leave a patient weakened by operation or disease sitting up in bed for more than a few hours at a time. A permanent sitting position, the legs being relaxed, is an invitation to thrombosis of the quiet type which so often causes embolism. It would be best that the patient about whom one is especially nervous should remain supine or even head downward until he is ready to begin getting out of bed. Indeed, unless the patient has a very vulnerable cardio-respiratory system, an elderly man subjected to prostatectomy had better be kept, during the post-operative period, head down and feet up for a good part of each twenty-four hours.

The prevention of increased intra-abdominal tension has been dealt with by Bancroft and his associates. They insist that abdominal wounds be so carefully closed that tight strapping and binders are not needed and they regard the prompt restoration of intestinal tone by the early use of semi-solid or solid food as essential. Probably the surgeon's handling of the abdominal viscera at the operating table is equally important. An adequate fluid balance, as Maddock and Collier have proved, merely requires an intelligent calculation. In these days, glucose (five per cent) and physiologic saline solution are given, intravenously for the most part, according to the particular indications.

Measures directed against thrombosis should start at the earliest possible moment, not several days after the exciting factor has appeared. This has been realized by Murray and Best in giving purified heparin after major operations in Toronto. By establishing, within an hour or two of operation, a continuous intravenous injection at an appropriate dosage, they raise the coagulation time to fifteen minutes, maintaining it there for several days, and have decidedly lowered, in a long series of cases, the incidence of thrombosis and em-

bolism The presence of heparin not only delays coagulation but prevents adhesion of the thrombocyte, that is, it completely abolishes thrombosis Unfortunately, the treatment is very expensive, and is certainly not yet available for general use One may perhaps depend upon Bancroft's clotting index as a measure of the threat of thrombosis, taking special precautions or using an anticoagulant drug for the case in which thrombosis seems imminent For an account of the test, Bancroft's writings should be consulted His formula is shown below

$$\frac{\text{Prothrombin (1)} + \text{Fibrinogen (0.5 to 0.7)}}{\text{Antithrombin (1)}} = 0.5-0.7$$

An index of over 1 points to a tendency to thrombosis and the need of preparing the patient by the use of a high protein, low fat and carbohydrate diet Whether or not this diet is given, sodium thiosulphate in ten per cent solution is administered intravenously for three consecutive days in a daily dose of ten ccm

*The Treatment of Established Thrombosis* must always hurry the return of blood from the legs and pelvis A solid thrombus, it must be supposed, occupies the upper femoral and more or less of the iliac vein But is a loose, detachable, propagating clot present at the proximal end of the thrombus? In the usual outspoken phlegmasia alba dolens it is very rarely present in the occasional quiet, barely noticeable and perhaps incompletely obstructing thrombophlebitis it may well be However, the only proof of its existence is the occurrence of a pulmonary infarction due to a nonfatal embolus Therefore, unless one believes that the common iliac vein or vena cava should be divided rather often on suspicion (1) the presence of the propagating process must be ignored and all efforts directed against its formation It isn't the patient's turning over in bed which should be blamed for the fatal embolism, but the presence of the detachable embolus, and

the best practical defense against the formation of the embolus is elevation of the lower limbs

Elevation of the legs has two objects the hurrying of a collateral stream past the proximal end of the thrombus, to prevent the growth of the fragile clot which a slow stream encourages, and the relief of edema. There is no reason why the leg should not be elevated and no reason against its being moved, the proximal end of the thrombus being within the pelvis and little influenced by such factors. Indeed, why worry about casual exercise when the patient must practice daily the athletic feat of using the bed pan? So the foot of the bed should be raised six inches and the swollen leg elevated still more upon an inclined plane or in a sling. A couple of pillows may be placed under the shoulders and head but the body should not be bent by raising the upper half of the adjustable bed. Under these conditions, the leg will have freedom of motion and should not be covered with ice bags. Beyond the fact that heat usually brings more comfort—if the thigh is painful—the ice bag delays the flow of blood rather than hastens it and occasionally inflicts a frost bite. The old custom was to sit the patient up in bed, apply ice to the flaccid legs and wait for swelling and fever to subside. Whether the existence of fever is actually an important consideration is unknown to the writer. When present, it may be expected to disappear with the edema. Under the system here described, as edema disappears, active exercise of the leg or legs in bed is begun and should be continued, with assistance if necessary, for some days before the patient is encouraged to get up. Then, with bandages applied up to the knees, he begins to walk, going to bed between his early attempts. From this time on, the use of the legs must slowly and regularly be increased. A return of edema is of course a sign of too much dependence and too little elevation. Standing, or sitting with the legs dependent, encourages swelling. Muscular exercise diminishes it.

Should pulmonary infarction occur, the question of dividing a great vein proximal to the thrombus comes up. For those who have not as yet been treated by elevating the lower half



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of the body, elevation should first be tried. But should infarction occur while the patient is being exposed to the favorable effect of elevation, the answer is difficult. It has already been explained that embolism from an outspoken, completely obstructing femoro iliac thrombophlebitis is unusual. It is far more common when swelling is slight or absent and a considerable current is able to flow past the thrombus. Should such a condition be found upon exploration at the groin, opening the common femoral—the current from the various entering branches being controlled—may permit extraction of the insecurely fixed clot by gentle suction. The femoral and its various branches should then be divided. However, the indications for this difficult procedure or even a direct attack upon the iliac vein within the pelvis are not yet clear. Approach to the left common iliac vein is best made transperitoneally, to the right, probably extraperitoneally from the direction of the iliac crest and right flank. But now the operator must determine the upper limit of the thrombus, which may be very soft. Unless he is fortunate enough to have divided the vein above it, he must still open the vessel and suck out the clot. Possibly the approach to all iliac thrombi had better be made, as in arterial embolism, through the common femoral.

**Thrombophlebitis in the Prostatic and Uterine Veins—**Thromboses found at autopsy in these vessels have indicated them to be a source of fatal pulmonary embolism, especially when no other source is discovered. Probably thrombosis in the plexus of veins draining the prostate or the uterus is able to progress through the hypogastric vein into the common iliac, obstructing the return from the leg and causing phlegmasia alba dolens. Indeed such must be an occasional cause of that disease. But may a long, nonobstructing, loose clot spring from a prostatic or uterine vein and may it break off leaving only the parent thrombus deep in the pelvis? That seems not to be known. There is certainly no way of identifying such a process during life. One can only hope that the routine treatment intended to forestall a femoro iliac thrombophlebitis will prevent its occurrence.

thrombosis is still active—is even perhaps extending—and that a propagating thrombus may well be growing up the femoral vein. There is all the clearer indication for femoral division in that experience has shown such treatment to cure the disease with remarkable rapidity. Of course it removes the danger of pulmonary embolism. One might suppose that division, even below the profunda femoris, would cause, for a moment at least, cyanosis of the foot. Such is not the case. The foot, which is usually a little cold, becomes warmer and even perhaps pinker than the other. In other words, a peripheral vasodilatation occurs.

Division of the femoral is performed under local infiltration with procaine or spinal anesthesia. A ten to twelve cm. (four to five inch) oblique incision parallel to the inguinal ligament and about three cm. (one inch plus) below it is satisfactory. The great saphenous vein is first found and is not disturbed. It actually is the best guide to the femoral. With retraction upward and downward upon the parts superficial to the aponeurosis, this layer is divided peripherally from the saphenous opening, exposing the common femoral just proximal to its division. Perhaps two cm. of the superficial femoral is isolated just distal to the profunda, silk ligatures are tied, the upper just below the profunda, and the vein is divided between them. This gives a good cuff both proximally and distally. If a segment is to be excised, a considerably longer exposure of the vein is needed. Should the propagating thrombus actually be encountered, it had better, as explained in the treatment of femoro iliac thrombophlebitis, be extracted by suction and the vessel divided. The wound is closed in layers with fine silk and requires only a local dressing.

Following femoral division or resection, the patient remains in bed, the foot of which is kept elevated four to six inches, for a week or for such a period as the surgeon feels is required for the healing of the wound. The thrombosis is no longer important. Bandaging the lower leg is advisable when walking is begun but a normal life can soon be resumed. In the writer's experience, cyanosis is not afterwards noticeable, but a little

tempted The difference between the two legs is usually very clear to the patient Not only is this test useful in diagnosis but in following the progress of the disease, for with healing of the thrombosis and the re establishment of a normal circulation, the sign disappears

*Treatment* varies with the stage at which the disease is first seen If the patient has but just complained of lameness and now for the first time exhibits a slightly swollen, bluish foot and soreness on forced dorsiflexion of the foot, conservative treatment should be tried The lower end of the bed is raised, on blocks, four to six inches above the upper, and the affected leg is placed on a soft pillow A large cradle is used to keep the bedclothes off the feet No restriction is placed on moving the leg, but on the other hand no attempt is made at this time to exercise it A couple of pillows may be placed under the head and shoulders If the patient is able easily to manage the bed pan for defecation he should do so If not, he had better get up once a day (applying a semi elastic bandage from toes to knee) and use the bedroom cabinet

Elevation in bed is maintained for at least ten days or until all signs of soreness on forced dorsiflexion of the foot and all edema have disappeared The next four or five days are spent gently exercising all the muscles of the leg in bed For it is held that such exercise should precede getting up and that a propagating thrombus need not at this time be feared Then the patient begins to get up, wearing, when he first walks, a semi elastic bandage from toes to knee He feels his way along, walking a little and again elevating the leg, gradually increasing the periods of use and shortening those of rest If no swelling or blueness of the foot is noticed, an active life is resumed, the whole period of treatment having been three or four weeks

If, on getting about, swelling and cyanosis recur or if the patient is first seen when he (or she) has already, during several weeks, undergone successive periods of elevation, apparent recovery, and a return of the original signs, the femoral vein is divided distal to the profunda For it is reasoned that

associated with a reddened, tender skin, local edema, and some induration. The redness is sometimes such as to suggest that a lymphangitis is present, and, moreover, the brownish discoloration which often afterwards remains does not always follow exactly the course of the vein. Perhaps the lymphangitis causes the thrombosis (Figure 25 shows how nearly the lymphatics are related to the superficial veins), in which case the common epidermophytosis of the feet may be a factor. In any case, such infection as is present rarely leads to suppuration. The process tends rather to become chronic, the thrombus remaining fixed in the vessel with little change over a period of several weeks. In the end, a combination of softening and organization occurs, by which the lumen of the vessel is restored, and it regains something very much resembling its previous state. But once having been thrombosed, a varicose vein is always liable to this accident, which is a sufficient reason for dealing radically with the process.

*Palliative Treatment*—There are two ways of making the thrombosis of varix last a long time: the first is to go about without an elastic bandage, the second is to go to bed, sitting up with the legs outstretched in a horizontal position. Both methods keep the venous stream slow and encourage continued thrombosis. By contrast, the process is made to disappear by applying an elastic bandage and leading an active life, or, rather more effectively, by elevating the feet above the head and so remaining in bed. The first of these last two methods is especially useful when thrombosis is confined to the lower leg so that an elastoplast bandage can firmly be applied from the toes to the knee, that is, up to a point well above the level of the process. Ten days of such treatment—elastic pressure hurries the venous stream—often causes the thrombus to disappear but of course leaves the way open for a recurrence. Elevation of the leg in bed acts almost more quickly and has the advantage that it is equally successful when thrombophlebitis is present in the thigh. Indeed, when the process has threatened to reach the saphenous opening, it is the only satisfactory treatment.



edema on hard usage of the leg may be present for some months. A brief note upon two typical cases of deep thrombosis in the lower leg will be found on page 216. A case in which femoral division was practised will be found below among a group illustrating diagnostic problems.

**Thrombophlebitis in Varicose Veins**—The fibrosed, unhealthy state of the varicose vein's wall, associated with a feeble or reversed current, is explanation enough of the common thrombosis of varix. One may presuppose infection in tissues of lowered resistance, or one may believe that the lining of a dilated, pocketed varicose vein actually cracks under heavy back pressure, since pressures of arterial height have been recorded on coughing or straining. The wonder is, not that thrombosis occurs sometimes, but that it does not occur always. It usually appears near the knee, more often below than above and in a prominent dilated vessel or pocket. From its point of origin it progresses erratically upward, as a rule, and for an uncertain distance, but once half way up the thigh it is likely to reach the saphenous opening. Beyond this it almost never goes, that is, it does not grow into the femoral either as a solid occluding thrombus or as a propagating clot threatening embolism.

The failure of thrombosis in a varicose vein to invade the femoral is a good example of the ending and healing of thrombosis where it encounters a strong blood stream. The writer knows of only two instances of pulmonary infarction caused by thrombophlebitis in varix. Both of these were successfully treated by high division of the great saphenous. Apparently the best reason for the solid attachment of a thrombus in the great saphenous vein is again the unhealthy state of the vein's wall. This is in accord with the general principle already laid down, that the more outspoken the thrombophlebitis the less the danger of embolism. The dependent position of the leg—most of the time—has little to do with the confinement of the process to the varicose vein, else the subject of thrombophlebitis had better never lie down.

The thrombophlebitis of varix is occasionally acute and as

sociated with a reddened, tender skin, local edema, and some induration. The redness is sometimes such as to suggest that a lymphangitis is present, and, moreover, the brownish discoloration which often afterwards remains does not always follow exactly the course of the vein. Perhaps the lymphangitis causes the thrombosis (Figure 25 shows how nearly the lymphatics are related to the superficial veins), in which case the common epidermophytosis of the feet may be a factor. In any case, such infection as is present rarely leads to suppuration. The process tends rather to become chronic, the thrombus remaining fixed in the vessel with little change over a period of several weeks. In the end, a combination of softening and organization occurs, by which the lumen of the vessel is restored, and it regains something very much resembling its previous state. But once having been thrombosed, a varicose vein is always liable to this accident, which is a sufficient reason for dealing radically with the process.

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Local applications are not essential. If any are used, heat is preferable to cold. It encourages hyperemia, brings comfort and presumably discourages further thrombosis, whereas the traditional ice bag delays the circulation, devitalizes the tissues, and encourages an extension of the thrombus. The only good which can be said of the ice bag is that it is often comforting, quite as much so as the hot-water bottle.

*Curative Treatment*—To check thrombosis, shorten the patient's disability, and prevent recurrence of the disease nothing can compare with resection of the great saphenous vein at its entrance into the femoral. The only contraindication to such treatment is the presence of a thrombus in the upper part of the vein. However, this contraindication is not absolute and in many cases it is difficult to decide whether or not thrombosis is actually present at the saphenous opening. Should it be determined beforehand, because of the presence of a thickened vein and local tenderness, that such is actually the case, the patient is subjected to the routine elevation of the foot of the bed. Within ten to fourteen days, the local thickening and tenderness will usually have disappeared, indicating that the thrombus has been organized or liquefied. As one follows a superficial thrombosis one is aware that the vein becomes continually softer, so that it is less and less easily palpated. From being a solid cord, the size of a lead pencil, it will often in a space of ten days, more or less, take on the character of a soft, barely palpable vessel. Its wall always becomes thickened, but not to such a degree as to suggest that the whole thrombus is organized. Apparently the most clot-like portion is liquefied and carried away.

Once the upper saphena magna is again open, it may well be resected. Opinion will naturally be divided as to when the operation should be performed, but the likelihood of recurrence is such that a high division had better be made sometime. Why not, therefore, resect the vein at once and avoid a subsequent hospitalization?

If exploration at the groin unexpectedly finds a thrombosed saphenous vein, it has been the writer's practice to pass two

ligatures under it at the point where it is exposed and divide the vessel between the ligatures. In so doing the operator should disturb it as little as possible but actually the danger of detaching a part of the thrombus is very slight. For not only is division made an inch from the femoral but a propagating clot very rarely grows into the latter vein.

Should the thrombus end proximally below the saphenous opening, the operation of dividing the great saphenous vein is performed in exactly the same way as for uncomplicated varix. One should have in mind, however, that almost every superficial thrombophlebitis is associated with some involvement of the perivenous lymphatics and some degree of lymphadenitis at the saphenous opening. Any suggestion of enlarged inflamed nodes is therefore a signal for especial care lest the glands be disturbed and spill infection into the wound. However, such an infection never seems actually to suppurate. Following high resection of the great saphenous, the thrombus clears up very rapidly. The varicose veins as well are usually cured.

**Thrombophlebitis in Nonvaricose Superficial Veins**—The form known as *phlebitis migrans* has been described in Chapter III as a complication of thrombo angitis obliterans. There it takes on its most typical appearance. However, a disease, seemingly in other respects identical, does occur in those who are not sufferers from Buerger's disease. In some of these there is a tendency to recurrence throughout life. Without any obvious cause, a stretch of vein, an inch or two in length, almost invariably upon the surface of the lower leg, becomes solid, thickened and slightly tender. In this state it remains for a week or two, and then, as it softens and apparently is restored to something very much like its normal condition, another area of thrombophlebitis, considerably proximal to the first, appears. The first may occupy the region of the ankle, the second, the upper calf, and a third, perhaps, the lower thigh. Apparently there is little tendency to embolism and continued use within an elastoplast bandage is about as successful as any other form of treatment. Whether the dis-

case would promptly disappear (on any one occasion) if the leg were subjected to continuous elevation is unknown to the writer. It is usually treated by only partial rest and elevation and has exhausted everyone's patience before it ceases to break out. A fairly typical example, in which high division was finally used, will be found included with several other case reports below.

The superficial thrombophlebitis which does not take the form of phlebitis migrans is actually more freakish and unaccountable than any other. It occurs most often perhaps in locally dilated veins which, however, are not a part of a varicose saphenous system. Local chafing, as in horseback riding, has been known to bring it on. Exposure to unusual cold, a severe bout of coughing, a trivial injury of any sort may occasion thrombosis. It seems to have the faculty of hanging on for considerable periods, and if not promptly cured may at any time quietly march out of a small radical into the main stem of the great saphenous and from somewhere in the course of that vessel give off an embolus. Altogether it is difficult to know whether or not to fear it. For in most cases such a thrombosis must heal with little to show for its presence. It must also be admitted that among adipose women, in whose fat a local thrombosis is not easy to identify, the diagnosis between it and a local lymphangitis or mild cellulitis must often remain doubtful.

*Treatment*—Following the general rule of treating thrombosis, the superficial form, if only suspected of being present, should be treated by elevation—of the legs above the head—over a period of perhaps ten days. This period is set to offer a safe margin for thrombophlebitis not easily palpated. As a matter of fact, the writer has seen it disappear completely from a vein near the ankle in less than a week. But if, as so often happens in this and other sorts, the thrombosis has already persisted for several weeks when first seen, it should receive more radical treatment. That is to say, the parent vein, usually the great saphenous, should be divided at the saphenous opening, after which the process is soon healed.

## Cases Illustrative of Various Sorts of Thrombophlebitis— and Their Treatment

The reader may, if he likes, come to a conclusion as to the nature of the disease in these cases and plan the treatment. He will perhaps prefer his own plan to that actually used.

Case 1—R C, an athletic man, forty eight years of age, while playing tennis six weeks before coming under observation, gave his left leg an unusual wrench. He suffered considerable pain but was able to continue play. He must have torn a muscle (?) in the upper calf for he noticed considerable local ecchymosis in the course of the days following. Nevertheless he kept about for three days, his lower leg somewhat swollen and painful. Then he took to his bed, with such relief that at the end of a week he thought himself fit to get about again. However, on getting up he soon noticed that the left ankle was swollen, the foot bluish. Again he went to bed, for nine days this time, and again on getting about the foot became cyanotic, the lower leg swollen.

When he came under observation, he had gone to bed for the third time and had had six days of it. The left leg looked normal but the foot felt cool to the touch as compared with the other and turned a little blue when hung for a couple of minutes out of bed. There was a slight feeling of soreness and tension, behind the knee, on forced dorsiflexion of the left foot. On the following morning, the patient applied a bandage and went to his office, but when seen that afternoon, the foot was blue and the marks of the bandage showed clearly upon the swollen leg.

*Diagnosis*—Rupture of muscle (and vein?) Thrombophlebitis of deep veins of left lower leg. Immediate division of femoral vein advised.

*Operation*—The femoral vein, which appeared normal and contained no thrombus, was divided just below the profunda. A segment excised was not remarkable. Immediately (on the operating table) the left foot, hitherto colder than the other, became equally warm and if anything a little pinker in color.

The patient left the hospital in a week wearing a semi elastic bandage In three weeks he had begun to exercise as usual At first the ankle would be found a little swollen each night but would be normal in the morning After six weeks he gave no further attention to his leg

Case 2—B S N, a woman, fifty two years of age, had suffered during her sixth pregnancy, nine years earlier, from an inflamed vein upon the inner side of her left thigh This troubled her during the last few months of her pregnancy During delivery (placenta previa) she lost much blood, and two weeks later developed a milk leg This came on with terrific pain and lasted for two months The leg had never been the same since, swelling at the ankle when she was for long hours on her feet

Four months after delivery and the onset of the milk leg, the patient suffered what seemed to be a pulmonary infarct (severe pain beneath the left breast but without hemoptysis) This was repeated, without obvious reason and in a far more severe form, eight years later—severe thoracic pain, fall of blood pressure and prostration Soon after recovery from this episode, the writer saw the patient and found a slightly darkened area upon the inner face of the upper left calf, the remains, he thought, of a recently healed great saphenous thrombosis No actual varicose veins As the patient had been up and about very little, he suggested seeing her again when she had been more on her feet but he only saw her six months later after her third and almost fatal pulmonary embolism

This embolism, which was marked by agonizing pain behind the sternum and a violent fall of blood pressure, was preceded only by a little patch of redness and soreness upon the inner side of the calf (in the region previously thought to be the scene of a saphenous thrombosis) This was remembered afterward In the meantime, the E K G was found to be consistent with a pulmonary embolism and the X ray of the thorax with a pulmonary infarct

Upon recovery, the state of the leg was the following Redness and soreness of the calf had disappeared. However,

higher up, on the thigh, a faint discolored patch over the course of the great saphenous vein was noticed. But at the same time there was typical discomfort back of the knee on forced dorsiflexion of the foot.

Thus the question was raised whether the last embolus had come from the saphenous system (evidence of recent thrombosis) or from the deep veins of the calf (dorsiflexion sign). More than this, there was, in the background, the old femoro-iliac thrombosis, so that the embolus could conceivably have come from the iliac region. It was decided to divide the great saphenous and to examine the femoral, with the idea of dividing it unless it contained a thrombus. It seemed desirable to operate promptly when the state of the vessels was likely to give a clue to the recent course of events.

At operation, the great saphenous was found whitish, thick walled, evidently the scene of an old thrombophlebitis (which might have been secondary to the original milk leg). There were enlarged, juicy lymph nodes about the saphenous opening as evidence of a recent process. The femoral artery and vein were embedded in fibrous tissue and were dissected apart with some difficulty (common result of old phlegmasia alba dolens). The vein contained no thrombus. It was divided distal to the profunda, between silk ligatures.

Immediate result, normal color and warmth of left foot. Normal healing. Unaccountable weakness of extensor muscles of both thigh, leg, and foot, from which recovery was gradual but complete. Ankle and foot afterwards swelled less than formerly. Last report, only six months after operation, showed continued good health. No one will ever know, of course, from which vessel the emboli had come, the great saphenous or a deep vein in the calf, but the episode is unlikely to be repeated.

Case 3.—E. D., a woman, sixty two years of age, the mother of two children. Typhoid fever, rheumatic fever, and malaria in youth. Following her first pregnancy, she had noticed enlarged veins on the inner face of the thigh and outer side of the left leg below the knee. No typical varicosity of the great saphenous system.



About a month before being seen by the writer, the patient noticed an area of redness and soreness over the enlarged veins outside and below the left knee, together with some crampy feeling in this region which came on suddenly at night and promptly disappeared. She kept about under considerable discomfort for four days, at the end of which time she consulted her physician who made a diagnosis of "phlebitis" and put her to bed. There she remained, sitting up most of the time, her leg on a pillow, for two weeks. The area of redness in the meantime subsided.

At the end of her two weeks in bed, the patient experienced a sudden epigastric pain and a desire to defecate (a common premonitory sign of pulmonary embolism). At stool, she was stricken with a sharp, severe pain in the right chest, becoming weak, breathless and apprehensive. With the aid of an oxygen tent and cardiac stimulants, she survived. As soon as possible, the left leg was elevated and the head lowered. Both E K G and X ray confirmed the diagnosis of pulmonary embolism and infarction.

On recovery from the embolism, the patient was found to present no asymmetry of her legs, no blueness or swelling of the left foot. The veins of the left leg lateral to the knee, were a little prominent as were those of the inner face of the thigh. No tenderness or masses, but at the groin, in the region of the saphenous opening, was a pencil shaped, slightly tender mass, three cm. in length which, from its situation, might have been either a thrombosed saphenous vein or a group of slightly inflamed lymph nodes. Under several days' observation this mass disappeared.

There was no discomfort on forced dorsiflexion of the foot.

*Diagnosis*—Superficial thrombosis, probably in the great saphenous vein (history, presence of enlarged veins and tender mass at saphenous opening) but possibly in the lesser saphenous (position of original soreness and redness).

*Operation*—Division of great and lesser saphenous veins. The lesser saphenous was found sclerosed and irregularly distended. Near the popliteal, it was small and not thrombosed.

It was not believed to have been the source of embolism but was divided.

The great saphenous vein at the groin was straight and thick walled. About a large branch, entering it laterally, the tissues were adherent. A vague mass of lymph nodes was present and was not disturbed. Evidence of recent inflammation in the upper saphenous and a large lateral branch being clear, and no thrombus being now palpable, the great saphenous was resected, from the femoral downward for over an inch (three cm.) Pathological examination revealed an organizing thrombus in the resected portion! A propagating thrombus must have grown from the great saphenous into the femoral while the patient reclined in bed for two weeks.

The patient made an excellent recovery. The left leg swelled a little at first at the ankle when the patient spent much time on her feet—actually less than before the operation.

Case 4—R E F, a man, forty five years of age, was first seen complaining of a sore spot upon the inner face of his calf, a little below the knee. Six years earlier he had noticed, at a time when he had been going up and down stairs a good deal, a sudden pain in the back of his left calf, which then became sore to the touch. He never really gave in to the illness which dragged along for six weeks, at the end of which time he experienced a sudden pleuritic pain and began to spit up blood. He was at first thought to have tuberculosis, a diagnosis afterwards contradicted by an expert on that disease. No one, however, had connected the state of the leg with that of the lung. He had since remained well until his present illness.

When seen by the writer three days after the appearance of the new area of soreness, there was noticed a little reddish streak overlying an obviously thrombosed vein. In those three days, the thrombosis had risen slightly until it reached the natural crease just below the knee, rather posteriorly. The leg was shaved and an elastoplast bandage applied from toes to knee. The patient was directed to go about as usual. Eight days later the region of the thrombosis was no longer sensitive and seemed to have healed but on the following day, the

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usually arises from a propagating thrombus occupying the whole length of the femoral, is rarely if ever survived. The embolus is too long and too heavy. However, it seemed wisest to cut off both sources. Resection of the diseased vein appears also to be actually beneficial to the peripheral circulation (release of vasospasm?)

Case 3 represents almost certainly a pure great saphenous embolism and brings up the question whether the propagating thrombus had floated, before detachment, in the common femoral or belonged entirely to the great saphenous system, having been detached from some point in the lower thigh or calf. The former seems the strongest probability in spite of the extreme rarity of embolism from the femoral end of the thrombosed great saphenous vein. That is, a propagating thrombus almost never forms and hangs in the strong femoral current when the saphenous vein is thrombosed. Here, however, the patient had remained in bed for two weeks, suffering from what must have been at first a very local superficial thrombosis, much of the time reclining and thus creating conditions favorable to propagation of a thrombus.

In Case 4, which should have been subjected to operation two months earlier, adhesive bandaging was given more than a fair trial. Apparently the state of the patient's vein was the important factor. He showed, according to Bancroft's index, no thrombophilic tendency, and his disease was quickly abolished by high division of the great saphenous. It is fairly typical of the phlebitis migrans type.

As a group, these cases illustrate the utter harmlessness, from the point of view of obstructing the venous return, of dividing a great vein when disease is present in its peripheral portion. The benefit to the circulation is perhaps due to the interruption of undesirable impulses passing along the vein, in which direction is not clear. The matter is perhaps analogous to arterial resection in the presence of a local plug. The cure of the thrombosis itself is always striking. After division or resection of the vein, the peripheral thrombophlebitis can almost be ignored.

bandage still being in place, a fresh area of thrombosis about two inches in length appeared just above its upper limit. Fresh adhesive bandages were therefore applied up to a higher level, but in a few more days, the thrombosing process jumped again, this time to the mid thigh. There it remained stationary, the lower areas being healed and free from soreness. The patient continued to use the adhesive bandage and during the following month, when in New York, consulted Dr. Bancroft who found his clotting index normal and advised high division of the great saphenous vein. Some three months after the onset of thrombosis, the process still being active, the patient consented to operation.

*The Diagnosis* was thrombophlebitis of the (nonvaricose) saphenous vein—phlebitis migrans type.

*Operation*—The great saphenous vein was resected in the usual way at its junction with the femoral. It was not diseased at this point. Several days later his rather badly diseased tonsils were removed.

The immediate result of operation was a rapid disappearance of the thrombophlebitis without further treatment. The patient has remained well.

### Comment

These four cases are presented, not to show that operation is the only treatment for thrombosis, but to illustrate how it may be used when a fatal embolism is feared. Case 1, a typical example of a persistent thrombophlebitis in the deep veins of the calf muscles, would have been treated, if seen early, by elevation alone. Yet after six weeks of shillyshallying, an operation offered not only the quickest, surest means of cure but an almost certain safeguard against embolism.

Case 2 was a complicated one, for it presented suggestive evidence of thrombosis in both the superficial system and the deep veins of the lower leg. Probably the embolisms which had already occurred came, however, from the great saphenous stem. This conclusion is reached principally because they were not fatal. Embolism from the deep peripheral veins, which

slightly enlarged arm is faintly cyanotic. It is impossible to say as yet whether such a condition is likely to persist. If so, an axillary periarterial sympathectomy or a sympathetic ramectomy would presumably be curative.

### PULMONARY EMBOLISM

In the foregoing account of thrombophlebitis, reference has repeatedly been made to the varieties most liable to the formation of an insecure propagating thrombus and the subsequent detachment of an embolus. A brief summary of these matters and a reference to promising methods of treating minor pulmonary embolism are included in the following paragraphs.

That there should be formed in a vein a floating, waving, friable clot of sufficient thickness and length to plug the pulmonary artery, it is necessary that a stretch of some ten to twelve inches in a medium to large sized vein should be available proximal to the thrombus. Such, of course, is found in the superficial femoral, the external, and the common iliac. A propagating clot may project from a vein of the deep calf or popliteal space into the femoral, from the femoral into the external and common iliac, from pelvic veins through the hypogastric into the common iliac and vena cava, and from the common iliac into the vena cava. Doubtless other situations might be suggested, but the femoral system and the veins of the pelvis, in both of which the venous stream can so readily be retarded, seem to be the most available sources of embolism. Particular spots anatomically favorable to thrombosis are present in certain parts of these vessels, namely in the upper part of the deep calf, the region of the groin, and the deep pelvis.

The large, fatal embolus is often a foot long and may show at one end the facet left by its detachment from the parent thrombus. Sometimes a number of separate emboli are found in the pulmonary artery or one of its main branches as if smaller fragments had been detached one after the other. Such great masses are usually rapidly fatal, but rarely the embolus

### Thrombosis (by Effort) of the Axillary Vein

This sort of thrombosis is rare and is evidently a very special form. It is seen almost always in active individuals, of the third, fourth, and fifth decades, who have recently made with one arm or the other—the right as a rule—some unaccustomed effort. Hence the name “thrombite par effort” bestowed upon it by the French. Those who have most carefully studied the accident believe that when the arm is abducted, and during expiratory engorgement of the axillary vein, the costo coracoid ligament indents the vessel (Lowenstem) or the subclavius muscle actually stretches the vein's wall at a certain point and injures a particular large valve (Gould and Patey). In any case, it is the arm most actively strained which exhibits the thrombosis. From the upper axillary vein, the process extends downward for a variable distance toward the elbow.

The usual history tells of some rather strained exertion with the arm elevated. Almost at once pain or swelling of the arm sets in, and during the next day or two the whole limb becomes edematous and somewhat blue. The superficial veins are apt to stand out, particularly over the shoulder. The axillary vein itself can be felt as a cord or elongated lump.

Treatment consists of rest in bed and elevation of the arm upon pillows. Fixation is unnecessary. Under these conditions the discomfort and swelling disappear very much more rapidly than is the case with a femoro iliac thrombosis. In ten days to two weeks, the arm will usually have regained its normal appearance, save perhaps for some enlargement of the veins about the shoulder. There may also be left some temporary stiffness—no permanent after effects. Embolism does not seem to be a danger.

As a rare complication of an effort thrombosis, the individual may be left, as in other forms of thrombosis, with some degree of vasospasm, even a mild causalgia like state. The writer has seen one such case. The individual suffers a pain, something like that of intermittent limp, on exertion, and the

haps a mild lymphadenitis at the saphenous opening. A deep thrombosis in some of the great plexuses among the muscles of the calf gives a characteristic story and often is betrayed by subjective discomfort behind the upper calf on forced passive dorsiflexion of the foot. The detection of a femoro iliac thrombophlebitis is less easy. The outspoken sort—phlegmasia alba dolens—causes the familiar painful or uncomfortable swelling of the whole lower limb, but this sort seldom causes embolism, and if it does, the embolus comes from the external or even the common iliac, a situation, which, except under the most unusual circumstances, must be regarded as out of reach. The quiet, incompletely obstructing, embolism threatening sort is practically undetectable—there may be no swelling or cyanosis and even no discomfort or tenderness over the femoral vessels at the groin. As for thrombosis in the depths of the pelvis, which is presumed to exist, it is absolutely silent.

An account of how these various processes can best be treated and how, in the presence of some of them, the patient can be protected against further embolism, has already been given. In many cases, effective preventive treatment can be established.

The treatment of the fatal type of pulmonary embolism by Trendelenburg's operation, performed upon the unconscious and seemingly moribund patient, that is, exposing the heart, opening the pulmonary artery and sucking out the embolus, should be studied in the writings of Trendelenburg and, more recently, of Nystrom. By a perfect cooperative technique, possible only in first rate hospitals, an occasional life can be saved, as Nystrom relates, yet a more promising field is probably offered by treatment designed to prevent thrombophlebitis, or, if thrombosis has already occurred, to cut down the incidence of embolism.

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will float for some days in the artery without being forced into one of its main stems or causing more than a partial obstruction of the pulmonary circulation. As a rule, however, the patient is struck down by breathlessness and an agonizing oppressive discomfort or pain in the mid line behind the sternum. Breathing is violent, the accessory muscles of respiration are called upon, and cyanosis is succeeded by pallor, feeble breathing and death. The first sign of trouble may be a vague abdominal discomfort and a desire to defecate. Occasionally the substernal pain and respiratory distress almost exactly imitate coronary infarction, so that only by the aid of an electrocardiogram can a distinction be made between the two.

Small emboli, causing pulmonary infarction, seem to be of two sorts: first, fragments of embolus detached from a large thrombus and, second, entire loose propagating clots from vessels of moderate size. The first sort are likely to be followed by the fatal detachment of a large embolus. The second cause only the familiar infarction, leaving an area of dullness and diminished respiration in one lung or the other and a patch of decreased aeration detectable by the X ray. As a rule, infarction causes the patient to expectorate some blood and a pleuritic pain is usually present. The seriousness of the situation depends upon the size of the embolus. Minor infarctions are recovered from very rapidly but the larger ones are shocking and require cardiac stimulation and the use of the oxygen tent. Naturally, the occurrence of infarction leads to a search for its source, in order to determine whether a vein can be divided proximal to the process, protecting the patient from further and perhaps fatal embolism.

The search for the source of any embolus will lead to a study of the superficial veins of the leg, the deep veins of the calf, the femorals and iliaes. The nature and course of thrombophlebitis in these various vessels has already been explained. Evidence of a thrombophlebitis in the great saphenous system will usually be found in the form of a story of local tenderness and redness, the actually palpable thrombosed vein and per

haps a mild lymphadenitis at the saphenous opening. A deep thrombosis in some of the great plexuses among the muscles of the calf gives a characteristic story and often is betrayed by subjective discomfort behind the upper calf on forced passive dorsiflexion of the foot. The detection of a femoro iliac thrombophlebitis is less easy. The outspoken sort—phlegmasia alba dolens—causes the familiar painful or uncomfortable swelling of the whole lower limb, but this sort seldom causes embolism, and if it does, the embolus comes from the external or even the common iliac, a situation, which, except under the most unusual circumstances, must be regarded as out of reach. The quiet, incompletely obstructing, embolism threatening sort is practically undetectable—there may be no swelling or cyanosis and even no discomfort or tenderness over the femoral vessels at the groin. As for thrombosis in the depths of the pelvis, which is presumed to exist, it is absolutely silent.

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## CHAPTER VII

### ARTERIAL ANEURYSM ABNORMAL ARTERIOVENOUS COMMUNICATIONS

#### ARTERIOVENOUS ANEURYSM AND FISTULA

##### ARTERIAL ANEURYSM

AN ANEURYSM is a local dilatation of an artery. It is the media which fails. This is the muscular layer of all but the largest arteries. Of these it is the heavy, elastic coat which confers such an astonishing combination of strength and elasticity upon the aorta and its main divisions. The media of the great arteries gives way chiefly because of syphilitic infections. Smaller vessels such as the femoral or popliteal suffer from aneurysm which is less apt to be syphilitic. Arteries often and actively plied, as at the knee and groin, have a greater tendency than others to crack or bulge, and syphilis as a background is not required. Of the great aneurysms, those of the aorta are the most common, those of the innominate and subclavian being next in rank. Of the peripheral aneurysms, the popliteal is the best example and as a matter of fact, by far the commonest.

The form of an aneurysm depends first upon whether a considerable stretch of arterial wall gives way or whether one particular spot weakens. A diffuse weakening makes a fusiform aneurysm, a local one, a saccular aneurysm. However, time modifies these forms. A saccular aneurysm, slowly enlarged, may so stretch the vessel, upon one side of which it first lay, that the original form of the artery is altogether lost. Then the artery, becoming flattened, may see its lateral opening lengthened and expanded until finally the sac assumes an almost fusiform shape. By contrast, a fusiform aneurysm may rupture, acquiring an almost saccular shape. Such distinctions

are important chiefly from the point of view of treatment. That is, a true fusiform aneurysm can rarely be subjected to a plastic operation intended to restore a lumen, but a saccular aneurysm can sometimes be treated by one of the ingenious procedures of Matas, "restorative" or "reconstructive" aneurysmorrhaphy. Some arterial dilatations are so extensive that the whole vessel widens over a very long distance. In the aorta this is common enough, but the same thing may happen to practically all the great arteries of the body. The vessels of the legs, for instance, rarely become arteriosclerotic winding channels one to two centimeters in diameter. Such states as that are not considered here. It is proposed only to describe the subclavian and popliteal types of aneurysm, as representatives of those which affect the circulation of the limbs, to give some account of the tests intended to reveal the nature of the collateral circulation and discuss very briefly the standard methods of treatment.

### SUBCLAVIAN ANEURYSM

This aneurysm, a representative of the great vessel type, is usually fusiform and presents itself as an expansile swelling which gradually appears above the clavicle at the root of the neck. If particularly large, it has been known to fill the space between the clavicle, scapula, and sternomastoid muscle, even lifting the clavicle and bulging into the axilla. It is more often right than left sided and nearly confined to males. Because of its proximity to the brachial plexus it is likely to be a cause of pain and weakness in the corresponding arm, and pressure upon the companion veins will result in venous congestion and edema. The radial pulse may be weak and delayed, the blood pressure in the corresponding arm lower than that of the opposite side. The natural course of the lesion is toward final rupture upon the surface at a point where the skin will already have become reddened and adherent. Before any surgical attack is made upon such an aneurysm, the state of the collateral circulation must first be studied. Actually there is no authoritative test for the efficiency of this circulation save by tem

## CHAPTER VII

### ARTERIAL ANEURYSM ABNORMAL ARTERIOVENOUS COMMUNICATIONS

#### ARTERIOVENOUS ANEURYSM AND FISTULA

##### ARTERIAL ANEURYSM

AN ANEURYSM is a local dilatation of an artery. It is the media which fails. This is the muscular layer of all but the largest arteries. Of these it is the heavy, elastic coat which confers such an astonishing combination of strength and elasticity upon the aorta and its main divisions. The media of the great arteries gives way chiefly because of syphilitic infections. Smaller vessels such as the femoral or popliteal suffer from aneurysm which is less apt to be syphilitic. Arteries often and actively plied, as at the knee and groin, have a greater tendency than others to crack or bulge, and syphilis as a background is not required. Of the great aneurysms, those of the aorta are the most common, those of the innominate and subclavian being next in rank. Of the peripheral aneurysms, the popliteal is the best example and as a matter of fact, by far the commonest.

The form of an aneurysm depends first upon whether a considerable stretch of arterial wall gives way or whether one particular spot weakens. A diffuse weakening makes a fusiform aneurysm; a local one, a saccular aneurysm. However, time modifies these forms. A saccular aneurysm, slowly enlarged, may so stretch the vessel, upon one side of which it first lay, that the original form of the artery is altogether lost. Then the artery, becoming flattened, may see its lateral opening lengthened and expanded until finally the sac assumes an almost fusiform shape. By contrast, a fusiform aneurysm may rupture, acquiring an almost saccular shape. Such distinctions

the afferent artery can be controlled. The same thing should, if possible, be done for the efferent vessel, to check any retrograde flow into the aneurysm from this source. It should then be possible to open the sac widely. Bleeding from any entering branches must be stopped by pressure with the finger, and the branches closed by silk or chromicized catgut stitches taken across their mouths. Next, with circular stitches which take a good grip upon the inner coats of the aneurysm, the afferent and efferent orifices are closed from within. By a series of similar circular stitches, placed at short intervals from one end of the sac to the other, the whole of a small aneurysm can then perhaps be obliterated, but if the sac is larger and friable, so that the stitches will not hold, the wall of the aneurysm had better be infolded and made into a compact mass by mattress stitches. Much will depend upon how strong a retrograde flow is found to enter the sac from any branches encountered. The less such a flow, the easier and less bloody the operation. This of course is the same general plan which Matas uses in the treatment of any aneurysm of a smaller vessel which can not be reconstructed or restored. In the end, the controlling rubber tubes are released and if the obliteration has been thorough, all bleeding will be found to have ceased.

### POPLITEAL ANEURYSM

This is decidedly the most common peripheral aneurysm, comprising perhaps fifty per cent of all such lesions, the femoral and femoro iliac (combined) coming next in frequency. It is usually of the fusiform type but may be saccular, representing what is almost a rupture of the artery at some one point. When still so small as to call no attention to itself by local swelling, the aneurysm sometimes causes coldness or numbness of the foot, perhaps an intermittent limp, that is, commonplace symptoms of an arterial deficiency. When it has grown larger, the actual tumor beneath the deep fascia of the popliteal space, which is so likely to interfere with the movements of the knee joint, often out of a clear sky calls atten-



porary occlusion of the afferent artery, since digital proximal compression, in the case of any but a small aneurysm of the third portion of the subclavian, is impossible. The clavicle must be removed, the proximal portion of the aneurysm exposed, and a Matas or Halsted aluminum band closed about the entering artery. If this is tightened just sufficiently to stop all pulsation in the aneurysm, the state of the circulation in the arm will become clear in the following day or two. In the event that the arm and hand remain reasonably warm and pink, nothing more, for the moment, need be done, but the state of the sac will of course be watched. If it becomes smaller, harder, and remains without pulsation, the aneurysm may be considered cured. If the response is unfavorable, that is, if the hand and arm become white and cold, threatening gangrene, the closed wound should be reopened and the band, which has injured neither intima nor media, should be loosened just sufficiently to restore the circulation. Even then, the development of the aneurysm may be checked or it may actually, by a process of thrombosis and organization in the periphery of the sac, be reduced in size. Naturally, its subsequent course should be followed with care. Even though the pulsation and enlargement recur, a collateral circulation will doubtless have developed.

*Operative Treatment*—Should the application of a band have failed to control the aneurysmal pulsation or should pulsation and swelling have reappeared after a temporary improvement, more radical treatment will have to be tried. This will usually mean some form of aneurysmorrhaphy, that is, infolding and obliteration of the sac, a method best adapted to preserving the collateral circulation.

For aneurysms of the great vessels, the obliterative aneurysmorrhaphy of Matas is most likely to succeed and produce a permanent cure. The region of the aneurysm is widely opened, the sac, with its afferent and efferent arteries, is as fully as possible exposed and the entering vessel especially is so well isolated that a piece of rubber tubing can be passed under it. Thus by lifting on the rubber tubing the current in

others, and those interested in the subject will do well to study his writings

The more simple yet reasonably reliable tests are the following

1 Delbet's test If the peripheral pulses distal to the aneurysm are absent, yet the limb is of good color and nutrition, the collateral circulation is almost certain to be dependable The reason for this is clear enough For if the main channels are obstructed or receive no arterial flow, the well nourished limb must be getting its blood by collateral channels

2 If the principal artery is compressed just proximal to the aneurysm and the peripheral parts fail to turn yellowish white but rather remain a reasonably pinkish white, the collateral circulation is sufficient

3 If the principal artery is compressed as above and an oscillometer or even a blood pressure cuff applied to the peripheral part reveals some trace of pulsation, the collateral circulation is probably sufficient

The more complicated and authoritative tests are the following

1. Matas's flushing test (attributed by him to Moszkowicz) which is especially useful in the case of a popliteal aneurysm but which can also be used for any femoral lesion which is not too high. The artery immediately proximal to the aneurysm being compressed by the fingers or a mechanical device until the pulsation in the sac is abolished, and the leg being elevated, an Esmarch bandage is firmly applied from the toes up to the lower pole of the aneurysm. In this way the leg is emptied of blood. It is kept in this state for ten minutes in young persons, but for not more than five in elderly Now, while pressure is maintained on the afferent artery, the Esmarch bandage is rapidly removed and any flushing (Reactive Hyperemia—Chapter I) must depend upon the efficiency of the collateral circulation It is very easy to follow the descent of the resulting blush. If the collateral circulation is very competent, the flush will reach the toes in a few seconds to a minute or so Usually it will go rather rapidly to the upper

tion to the expansile pulsating swelling. If the sac cracks open at some point, the aneurysm may enlarge rather rapidly by a process of giving way, thrombosis, organization, and further stretching or rupture. If the peroneal nerve is injured by pressure there will be some degree of toe- or foot drop. Swelling and cyanosis of the foot and ankle are to be expected, but the most serious complications come from thrombosis within the sac. If this process extends into the efferent arteries, the anterior tibial and the terminal branches, that is, the peroneal and posterior tibial, the foot will occasionally become gangrenous, especially in arteriosclerotics whose collateral circulation has not become abundant.

Most popliteal aneurysms occur in middle life and for no apparently sufficient reason, though forced flexion followed by a violent muscular effort has sometimes been observed and syphilis is present in a small proportion of cases. They are almost unknown in women, even though elderly females often suffer from arteriosclerotic disease of the arteries in their lower limbs. The truth is that active use of the legs soon after the arteries have begun to stiffen rather than advanced arteriosclerosis seems to be the cause of most popliteal aneurysms. Once the expansile pulsation of a good sized popliteal aneurysm is present, the diagnosis is clear. At an early stage, however, when only signs of arterial deficiency are evident, diagnosis is very difficult. The aneurysmal sac may be small or sufficiently thrombosed to prevent a deep pulsation from being appreciated, that is, if palpable at all, it will feel solid. Yet all the while it may be responsible for a serious intermittent limp or impending gangrene. This calls attention to the combination of very low peripheral skin temperatures with an excessive elevation of the oscillometer readings at the knee. In case of doubt arteriography can of course be used.

**Tests of the Collateral Circulation**—It is in the case of the aneurysms of the lower limbs that special tests of the collateral circulation are particularly needed. The upper limb, in this respect, is far better safeguarded. Matas has made a very thorough study of these methods, condemning some, accepting

tubing the sac is opened from end to end and cleared of thrombi. All entering vessels are closed by stitches taken through the inner coats of the aneurysm. The sac is then closed from within by a series of circular stitches. If it is too large or too friable for such a step, it is infolded and matted

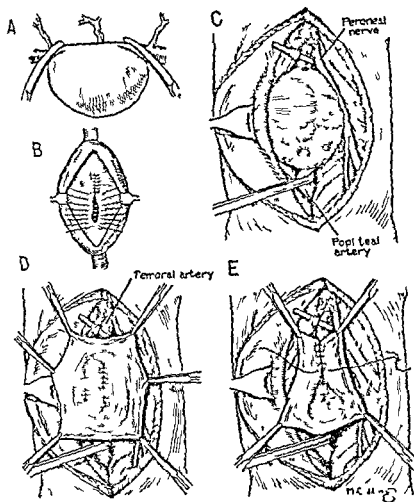


FIGURE 23 ANEURYSMORRHAPHY—after Matas. *A* After Matas's sketch showing how most of collateral circulation can be controlled *B* Matas's plan of obliterative aneurysmorrhaphy *C* A popliteal aneurysm, showing the sac exposed (the peroneal nerve has been compressed) *D* The sac partly closed by aneurysmorrhaphy *E* Further closure of sac with silk. The free edges may now be turned in and united by mattress sutures

level of the ankle, after which it spreads to the toes in a hesitating, erratic, and patchy manner. However, if the toes are colored pink in three minutes, the result may be considered satisfactory.

Should the flushing time be considered too slow, the patient's leg should be subjected to measures intended to improve the collateral circulation. These will include compressing the afferent artery for perhaps ten minutes at a stretch several times a day, and the usual vasodilating stimuli, especially the application of heat to the body. Vasodilating exercises might cause rupture or thrombosis in the aneurysm. Too much heat applied to the limb might be harmful though the leg should always be kept warm. Massage, avoiding the sac itself, will be useful.

2 The Application of a Matas or Halsted aluminum band—The rationale of this test has already been explained. The band should not injure the afferent artery and should be placed so close to the aneurysm as not to blot out any collateral arteries however small. The various possible results of its application have been described under Subclavian Aneurysm. If the peripheral part remains well nourished, the band may be left on with a reasonable hope of cure. If the peripheral part does not seem viable, the band is loosened a trifle and the case followed, in the hope that further treatment will not be needed and with the feeling that a collateral circulation is being developed.

If the pulsation in the sac is not controlled, some other procedure to cure the aneurysm is required.

**Surgical Treatment of Popliteal Aneurysm**—Undoubtedly the routine method used should be Matas's *Obliterative Aneurysmorrhaphy*, but for those skilled in the treatment of aneurysm, *Restorative* and *Reconstructive Aneurysmorrhaphy* are available and are occasionally useful.

*Obliterative Aneurysmorrhaphy*—This operation, already described under Subclavian Aneurysm, is pictured in Figure 23. The afferent and efferent vessels having been brought under control, preferably by elevation upon a piece of rubber

of the vascular malformations and arteriovenous fistulas have the appearance of innocent superficial swellings, others take the form of large superficial vessels much like varicose veins, and yet others are pulsating masses, which call attention to themselves by a thrill and audible bruit, and are evidently dilated by receiving an arterial stream. At first sight, the relation between these various types is not clear, but basically all such as are not of traumatic origin belong to one family, that is, they are failures of development in the common capillary plexus of the embryo from which the arteries and veins are evolved. All are apt to be called angiomas, or hemangiomas, with the implication that they grow, and actually there are tumors, some of them malignant, which originate in vascular endothelium. However, the arteriovenous malformations rarely grow in the sense that their parts multiply. They enlarge because they swell as a result of dilation of the blood spaces or vessels of which they are formed. The simplest of them is a swelling composed of undifferentiated spaces, neither arterial or venous. Such is the "naevus", which may be "capillary" or "cavernous" and which may have the purplish covering of the port wine stain, a coat of normal skin, or a mixture of both. By contrast, the arteriovenous fistulas are serious and occasionally terrifying lesions. The great arteries and veins are formed, yet retain connections from one to the other, so that at one or many places blood pours from artery into vein, causing obvious dilatation and carrying in its train some very remarkable changes, both in the part involved and in the circulation in general. In other words, it makes a great deal of difference whether the malformation occurs in the vascular bed of the skin and subcutaneous tissue or in the great vessels serving a limb.

Both the capillary and cavernous types of malformation are supplied with arterial blood but so indirectly and with so little force that they never pulsate—the tissue merely resembles a very vascular sponge with smaller or larger meshes. Another type has a more direct connection, usually by a series of tiny vessels, but there need be no actual pulsation in the

together by mattress sutures. The controlling tubing at either end of the aneurysm can be loosened from time to time to see if hemostasis is satisfactory.

*Restorative Endoaneurysmorrhaphy*—This operation is only possible in saccular aneurysms, especially when the original arterial lumen is well preserved, so that even if the lateral opening is large, the original channel is present as a groove. The blood supply being controlled by tubing or rubber covered artery clamps and the sac laid widely open, the slit or gap in the side of the artery is closed with a continuous stitch of fine oiled silk reinforced with a number of individual silk or chromicized catgut stitches. The current is then allowed to resume its natural course. It must of course be determined that the efferent artery is not thrombosed.

*Reconstructive Endoaneurysmorrhaphy*—This is the aneurysmal expert's dream which is almost never realized. There is seldom any real need of the operation, which in any case is only possible of performance when there is present, from one end of the aneurysm to the other, a straight posterior lane of normal intima. The walls of the aneurysm must then be sewed together in such a way as to leave a posterior channel. Mats recommends that this channel be constructed over a piece of rubber tubing which is extracted before the long row of stitches, carefully placed, is finally tied.

Should syphilis become rare or extinct, the arterial aneurysms of the lower limbs will be practically the only ones encountered. Even today, many a surgeon goes through life without seeing any of them. Probably the arteriovenous sorts are better worth studying.

#### ABNORMAL ARTERIOVENOUS COMMUNICATIONS

There is here presented a brief account of such unnatural arteriovenous connections, both congenital and traumatic, as are likely to appear upon the extremities, with the idea of sorting out the different varieties and distinguishing these uncommon and often serious lesions from the more familiar and harmless states which in some degree they resemble. Many

of the vascular malformations and arteriovenous fistulas have the appearance of innocent superficial swellings, others take the form of large superficial vessels much like varicose veins, and yet others are pulsating masses, which call attention to themselves by a thrill and audible bruit, and are evidently dilated by receiving an arterial stream. At first sight, the relation between these various types is not clear, but basically all such as are not of traumatic origin belong to one family, that is, they are failures of development in the common capillary plexus of the embryo from which the arteries and veins are evolved. All are apt to be called angiomas, or hemangiomas, with the implication that they grow, and actually there are tumors, some of them malignant, which originate in vascular endothelium. However, the arteriovenous malformations rarely grow in the sense that their parts multiply. They enlarge because they swell as a result of dilation of the blood spaces or vessels of which they are formed. The simplest of them is a swelling composed of undifferentiated spaces, neither arterial or venous. Such is the "naevus", which may be "capillary" or "cavernous" and which may have the purplish covering of the port wine stain, a coat of normal skin, or a mixture of both. By contrast, the arteriovenous fistulas are serious and occasionally terrifying lesions. The great arteries and veins are formed, yet retain connections from one to the other, so that at one or many places blood pours from artery into vein, causing obvious dilatation and carrying in its train some very remarkable changes, both in the part involved and in the circulation in general. In other words, it makes a great deal of difference whether the malformation occurs in the vascular bed of the skin and subcutaneous tissue or in the great vessels serving a limb.

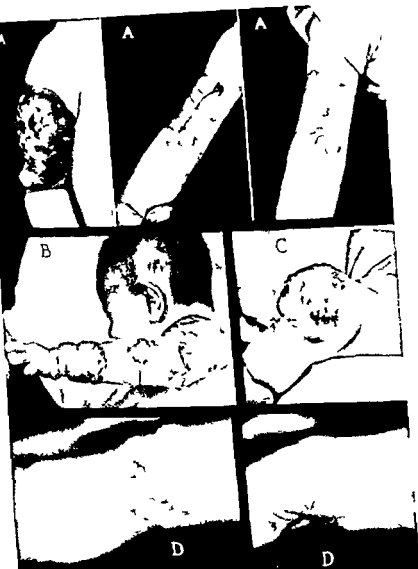
Both the capillary and cavernous types of malformation are supplied with arterial blood but so indirectly and with so little force that they never pulsate—the tissue merely resembles a very vascular sponge with smaller or larger meshes. Another type has a more direct connection, usually by a series of tiny vessels, but there need be no actual pulsation in the



receiving veins nor any bruit or thrill The connection may indeed be so insignificant that the blood of the prominent veins which mark the lesion is merely given an arterial tint and a rather high oxygen content Occasionally it happens that one of these relatively quiet forms is injured or undergoes some other unexpected change by which the afferent artery is able to pour a large stream *directly* into it Then the original angioma and the efferent veins dilate and pulsate, forming what has often been called a cirroid aneurysm This is most apt to occur upon the scalp

The arteriovenous aneurysm or fistula is especially common at the root of the neck or of a limb, because in these situations great arteries and veins lie rather superficially in close contact and are held together in a fibrous sheath. If they are malformed, that is, not fully differentiated from one another, there may be multiple fistulas between them, a series of small channels making connections over a considerable distance, as, for instance, for the length of a thigh, or even through a good part of the arm and forearm A fistula has usually been defined as a direct opening or simple narrow passage, whereas an aneurysm is held to be a sacculaton interposed between the artery and vein The cirroid aneurysm then is merely one which presents a varicose appearance Such distinctions, even if valid, are of no basic consequence

The traumatic arteriovenous fistula differs in having only one connection (unless one artery communicates with two veins) and that usually a relatively large one such as may be made by flying glass or steel, by the stab of a narrow blade or a bullet wound Some of the aneurysms of the ancients were probably of this kind and must have been made by spears or arrows However, pure arterial aneurysms must also have occurred even though syphilis was apparently unknown before the end of the fifteenth century Thus the operation of Antyllus—ligation of the afferent and efferent vessels and removal of the sac—which has been handed down as a classical procedure, may have been used for lesions of either sort A common site for the acquired fistula in these days is the region



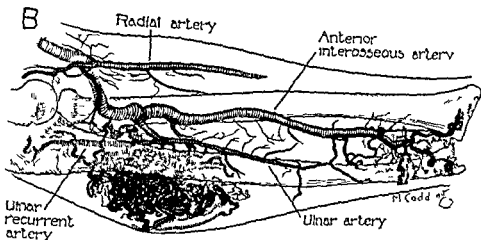
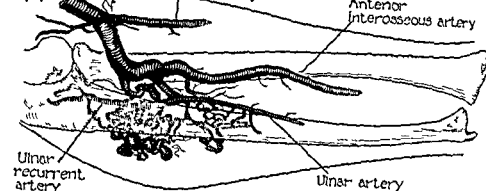
ABNORMAL ARTERIOVENOUS COMMUNICATIONS I Cavernous angioma Treatment by use of carbon dioxide now followed by partial excision (courtesy of Dr D W MacCollum Children's Hospital Boston) B Extensive cavernous hemangioma in process of treatment by carbon dioxide now C Prominent but localized cavernous hemangioma treated by excision (courtesy of Dr Robert F Green Children's Hospital Boston) D A seemingly innocent capillary nevus The infra red photograph on the right shows how large were the veins with which it was connected (courtesy of Dr George D Cutler Children's Hospital Boston)



ABNORMAL ARTERIOVENOUS COMMUNICATION. A huge vein fed by very small arteries—as associated with a capillary nevus of front of thigh. Treated by excision, and by the subsequent injection of sclerosing solutions. Note subcutaneous hemorrhage in the postoperative photograph. G.C. 15913.



ARTERIOVENOUS FISTULA CONGENITAL J.J.M. 51948 a man aged nineteen  
Infra red photograph before operation See arteriogram Plate VIII, A and B



J. M., 51518. Same patient as shown in Plate VII. Sketch after arteriograms made following the first operation in which only the proximal of the fistula was divided. *A* First exposure. Proximal connections with contorted mass of veins on back of arm. *B* Second exposure (1 second later). The radial and ulnar arteries free from fistula. are not filled by the thrombotic material. The fistula are chiefly from the ulnar recurrent and anterior interosseous arteries. The flow follows the course of least resistance, that is, through the various fistulas.



C AN ARTERIOVENOUS ANEURYSM OF THE BRACHIAL ARTERY—following a wound by a fragment of metal. A case treated and published by Drs. Leach and McGuire who have kindly permitted a reproduction of their illustration (Courtesy of *Annals of Surgery*, 109:643 Oct., 1938)

behind or above the clavicle, less frequently the groin, and the bullet is perhaps the most common cause. The nature of the great mass of pulsating veins which soon develops and of the changes in the heart which so often follow will be explained in a subsequent account of the lesion.

### HEMANGIOMA CAPILLARY AND CAVERNOUS

The familiar and picturesque deep purple birthmark upon the face or scalp, which so accurately occupies the field of one of the *trifacial* branches is a rather pure hemangioma, very superficial and capillary or deep and cavernous as the case may be. Not all congenital malformations are so clean cut. Some subcutaneous swellings are unclassifiable, being disorders of the subcutaneous structures in general, lymphatic as well as arteriovenous, as is told in the following chapter. The hemangiomas as a rule are compressible, that is, much of their content can be expelled by pressure and returns on release. No vessels are seen entering or leaving them, nor do they pulsate. Such as these occasionally appear on the limbs. They are usually raised above the surface and sharply marked off from the normal surrounding skin. Their covering may be wholly of the purple capillary sort or of a reasonably normal skin. But there is always some admixture of port wine stain in the form of smaller or larger patches. Multiple lesions are sometimes seen. Several of these malformations, all of the cavernous type, with a more or less capillary surface are shown in Plate X. These are taken from the records of the Children's Hospital in Boston, to the staff of which the writer is much indebted.

*Treatment* is usually surgical excision. Whereas on the face or scalp the large hemangioma must often be treated by the application of carbon dioxide snow, by multiple punctures with the endothermy needle or perhaps by radium or the X ray, it is seldom that those of the limbs can not completely be excised. They may be fed by several large vessels, but since the operation can be kept in the field of normal tissue about them, the control of their blood supply is not too difficult. The

muscular aponeurosis is apt to be missing, so that the base of the angioma must be dissected from the underlying muscle. The lesions shown in Plate X, have been treated by various methods or combinations of methods as is told in the legends.

**Tumor of the Cutaneous Glomus Glomangioma.**—This rather rare tumor is especially worth having in mind because it has the peculiar quality of being extremely sensitive and painful. Long known as "subcutaneous painful tubercle" it was first shown by Masson (1924) to represent an abnormal development, that is, a hemangioma, of the cutaneous glomus, the widely distributed tiny neurovascular organ which governs the rapid vasomotor reactions of the extremities. By its means, the arteriovenous flushing passages in the deep skin of the hands and feet are opened (Chapter I) raising the temperature of the surface. In the upper limb, the glomus tumor is most often found upon the fingers, the thenar and hypothenar eminences, and especially beneath the nails. In the lower, it is more erratically distributed. Rarely it is seen upon the body.

Attention is called to the glomangioma by a sensitive spot which causes the individual to protect it against all contacts, whether by friction or pressure. Pain of a knife like, radiating sort, is also excited by cold and is even spontaneous, but for all this sensitiveness Bailey remarks that most patients harbor the tumor for many years before seeking relief. Over it, the skin may be normal in color and without elevation, or may be bluish and elevated. Under the nail it makes a purplish spot. Actually, the glomangioma is so very vascular that, when exposed, it must display a color somewhere between red and blue. Its size is small, rarely more than a centimeter (one fourth to one half inch) in diameter and it does not grow, having no malignant tendency whatever.

Pathologically, the tumor is distinguished by showing certain epithelial glomus cells, a considerable admixture of fine nerve fibers and structures reminiscent of the contorted vessels of the typical glomus.

A very interesting feature, first noticed by Barre, for

which watch should be kept, is the association of the glomangoma with a chronic sort of vasomotor change in the direction of heat and flushing (or coldness and cyanosis) of the extremity

*Treatment* is surgical excision, which offers no especial difficulty and is entirely curative. A nerve block with procaine should be used wherever possible, rather than local infiltration

### CONGENITAL ARTERIOVENOUS FISTULA OR ANEURYSM

The appearance of this lesion varies enormously according to the nature and extent of the connection between artery and vein. There will perhaps have been noticed from birth a group of veins upon some part of a limb, a patch easily distinguished from the area about it and without visible superficial afferent or efferent vessels. Often the skin over some parts or all of the patch will be more or less abnormal in appearance, a little purplish or brownish. Sometimes a long dilated vein or plexus of veins will pass down an extremity. Along its course the skin will show blotches or spots of discoloration and appear slightly pitted or irregularly bossed. Usually such a lesion will take roughly the form of a broad band or occupy part of a foot or hand following in an erratic way one or more dermatomes.\* In other words, although large veins may be present, there is apt also to be a suggestion of the port wine stain, the capillary nevus.

A mass of veins or a single vein of this kind does not pulsate. Indeed, if its anatomical situation is such as to correspond to the familiar saphenous varicosity, it may be mistaken for varix. There is no swelling of the limb but there will occasionally be lengthening of some bone. The writer has seen a rather local lesion of this sort, chiefly confined to the outer side of the lower leg, external malleolus, and dorsum of the

The dermatomes upon the extremities correspond to the sensory zones upon the body which take their origin from the spinal segments. Those of the arms and legs are elongated and have no representation upon the body. Their number corresponds to that of the nerve roots which furnish the nerve supply respectively for the upper and lower limbs.



foot, which led to lengthening of the leg at one period of the child's growth, though subsequently the two limbs became equalized. The skin over such an angioma will perhaps feel warm to the touch as compared with other parts. It is impossible, without a study of the blood drawn from the distended vein, to determine the size of the arterial fistula. In the absence of a bruit, the arterial connection can only be very indirect, yet it may be sufficient to cause considerable difficulty in controlling bleeding both at the time of excision, if a surgical operation is used, or afterwards, as the following case shows.

G C, a young man twenty one years of age, had noticed all his life a dark patch of skin upon his left thigh and a large vein which seemingly emerged from the muscle on the upper, external surface of the thigh near the gluteal fold and ran down nearly to the ankle. By the Trendelenburg test, this vein lacked valves. It was in fact varicose. The lower limbs were otherwise symmetrical. No pulsation could be felt and, apparently, before surgical treatment was undertaken no bruit was searched for, yet subsequently a faint and utterly nonrhythmic, bubbling or clicking noise was heard through the stethoscope over the discolored area of skin above the knee. Blood from the vein was unfortunately not examined.

Through a long incision in the thigh and another in the lower leg the greater part of the vein was excised. The vascularity of the tissues was noted and at one or two points, notably where the vessel emerged from an opening in the fascia lata, bleeding was difficult to control. Throughout its course in the thigh the vein gave off a series of "infinitely small branches apparently connecting with the naevus" which lay principally median to it. Healing was complicated by an accumulation of blood clot in several parts of the wound, so that the patient was unable to leave the hospital for six weeks.

Ten months later G C returned for the treatment of an ulcer and shallow sinus at the upper end of the wound in the thigh. Serious bleeding had occurred from this point on several occasions. Upon excision of this area a plexus of large

veins was found, lying partly upon and partly beneath the fascia lata. Painstaking removal of the plexus was followed by permanent healing. Three years later, various dilated tortuous veins in the lower leg were successfully treated by the injection of sclerosing solutions and at the present time, sixteen years after the original operation, the leg gives no further trouble. It looks very much as it did in the photographs taken immediately after the first operation (Plate XI).

Cases much like the above have many times been described. In one of those recently published by De Takats, who has very kindly permitted me to reproduce several of his sketches, an additional and very troublesome feature was the presence in the birthmark of very delicate multiple capillary angiomas

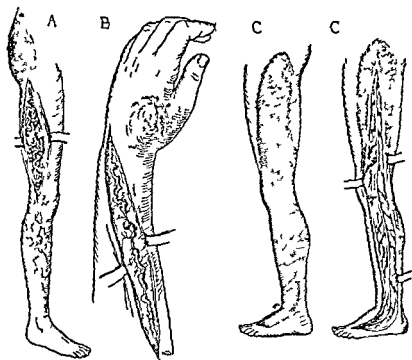


FIGURE 24 ABNORMAL ARTERIOVENOUS COMMUNICATIONS (By courtesy of Dr de Takats. Published in *Surgery Gynecology and Obstetrics*, 55:227-233 (Aug.) 1932. Figures 6, 8, and 12.) These bring out the associated capillary nevi and, in one case C, peculiar spots consisting of delicate collections of vessels which bleed easily.

which bled readily. By such lesions the nutrition of the extremity is in no way harmed, the circulation in general is not affected, and only if the angioma chances to increase the blood supply of the growing end of a bone or bones will any effect upon development be observed, that is, the extremity may be overgrown. A very different sort of disorder is caused by injury to the hemangioma. Then dangerous external bleeding may occur, or, as already explained, if the connection of an efferent artery with the lesion is suddenly widened, a bruit or thrill or even a palpable pulsation may develop. The distant results of such a change require separate description.

### **The Physiological Changes Consequent upon Large Arteriovenous Fistulas or Aneurysms**

These changes have long excited the curiosity of those who have been obliged to deal with such disorders. For a historical account of the many fundamental observations upon this subject, Holman's monograph should be consulted. In this country, Matas, Halsted, Reid, and Holman, in particular, have made significant contributions both to the study of the lesion as well as to its treatment. Most of the observed clinical findings have been reproduced experimentally by Holman. It appears that the secondary effects, namely, dilatation of the afferent artery and of the heart, are proportional to the size of the fistula. Considerable time is required for their development and this again is related to the size of the unnatural connection. Moreover, the nearer the heart the fistula is situated the more rapid and extensive will be the secondary changes. With such modifying circumstances in mind, the following may be considered to represent the usual result of the serious arteriovenous communication. The explanations given are the result of much experiment and observation, but need not necessarily be considered correct.

*General Effects upon the Heart and Circulation*—A large fistula calls for unnaturally rapid and vigorous contractions of the heart, increases the cardiac output, causes dilatation and perhaps hypertrophy of the heart, and may in time lead

to decompensation. When arterial blood pours from a large artery into a vein, the circulation in the limb, or neck, or organ, as the case may be, is short circuited. An easy path is made, by which the normal, resistant vascular bed of small vessels and capillaries is avoided. As between the distal capillary bed and the fistula, the stream therefore chooses the fistula. The sudden flooding of the capacious venous system which necessarily occurs has the effect of a massive hemorrhage. The arterial pressure falls and, in consequence, the heart beats faster. Venous pressure is elevated. Sufficient blood now being supplied to the right side of the heart, more blood is put out per beat and the arterial pressure rises. In this way the systolic pressure may gradually be restored to a normal figure but the diastolic pressure remains low, the pulse pressure being increased much as an aortic regurgitation (Lewis and Drury). The tendency of a fistula, then, is first to lower and then raise blood pressure, raise the pulse pressure and increase the pulse rate.

To compensate for the loss of blood on the arterial side, which results from flooding the veins, an increase in blood volume occurs,\* and the cardiac output becomes greater. Yet the fistula continues to carry much of the larger volume into the shorter circuit and so, to accommodate the increased bulk of blood, the heart and blood vessels dilate. Only the large fistulas induce this change and especially those not too far out toward the periphery. A fistula between the external iliac artery and vein is more potent than one in Hunter's canal.

The converse of these changes has repeatedly been demonstrated. If the fistula is temporarily closed by pressure, the heart rate is slowed, the blood pressure rises. When the fistula is permanently closed, the blood volume is soon reduced, the blood pressure, temporarily elevated, again falls, and the size of the heart is restored to normal.

This Holman maintains on what appear to be satisfactory experimental and clinical grounds. Reid is inclined to disagree. However no one disputes the fact that the heart rate is increased, that the heart puts out more blood with each beat and that the heart tends to dilate.

which bled readily. By such lesions the nutrition of the extremity is in no way harmed, the circulation in general is not affected, and only if the angioma chanced to increase the blood supply of the growing end of a bone or bones will any effect upon development be observed, that is, the extremity may be overgrown. A very different sort of disorder is caused by injury to the hemangioma. Then dangerous external bleeding may occur, or, as already explained, if the connection of an efferent artery with the lesion is suddenly widened, a bruit or thrill or even a palpable pulsation may develop. The distant results of such a change require separate description.

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*General Effects upon the Heart and Circulation*—A large fistula calls for unnaturally rapid and vigorous contractions of the heart, increases the cardiac output, causes dilatation and perhaps hypertrophy of the heart, and may in time lead

*The Progress of an Arteriovenous Fistula*—A small fistula is very occasionally closed by thrombosis and organization, one of modest size often remains stationary, but a large one tends to become larger. As the afferent artery and efferent veins dilate, there is a natural and purely mechanical tendency to widening of the fistulous opening. As the opening widens, peripheral resistance diminishes, more blood passes through it, the total volume of blood increases, the heart and proximal arteries dilate further, and so the fistula enlarges still more, a true vicious circle. If, however, at any time, pressure in the receiving veins rises enough to equal the peripheral resistance beyond the false passage, dilatation of the short-circuiting fistula will cease, and the vicious circle will be broken. By this same equalization, the dilatation of the heart is brought to an end, and ultimate degeneration of that organ may be prevented. Actually, in a young individual, the increased blood volume and elevated cardiac output are compatible, even over many years, with a heart of normal size and behavior.

### Summary of the Effects of an Arteriovenous Fistula

- 1 The effects are greater according as the fistula is (a) larger and (b) nearer the heart
- 2 The arterial blood pressure falls
- 3 The pulse rate is increased
- 4 The venous pressure rises (between fistula and heart and perhaps elsewhere)
- 5 The blood volume is increased
- 6 The cardiac output is increased
- 7 The heart becomes dilated (and perhaps hypertrophied)
- 8 The afferent artery becomes dilated and weakened
- 9 The efferent veins become dilated and strengthened
- 10 The collateral circulation about the fistula to peripheral parts develops
- 11 The limb may become overgrown but its terminal part may become ill nourished
- 12 All these effects are reversible. If the afferent and efferent vessels are divided and the fistula removed, the peripheral part is well nourished by the collateral circulation. Indeed nutrition of the peripheral part will be improved.

*Local Effects upon the Blood Vessels and the Peripheral Parts*—The tendency of a fistula is toward the "venafication" of the proximal artery, toward "arterialization" of the receiving vein and toward the establishment of an abundant collateral circulation. Reid points out that under the influence of the lowered resistance of the fistula and the lack of recoil against the arterial wall, the latter seems to undergo a sort of degeneration. Its muscular and elastic fibers deteriorate and it soon dilates. Possibly, as Holman suggests, the greatly increased blood mass of the shortened circuit enters into this dilatation. In any case, the process is often carried well back toward the heart and very rarely occurs distal to the fistula. It may be so serious that after excision or repair of the fistula, the afferent artery becomes an aneurysm or actually ruptures. The receiving vein, by contrast, not only dilates but hypertrophies. All coats are thickened and strengthened, the elastic material being greatly increased. This results in one of the most conspicuous features of the large fistula, the great mass of dilated, pulsating veins.

The effect of this local change upon the peripheral part of the limb, in the region of and for some distance beyond the false opening, is to enlarge it and to make its surface unduly warm. Peripheral to this swollen, hot area, the extremity is left cool and sometimes actually ill nourished, so that local necrosis and ulceration may occur. Strangely enough, however, a collateral circulation of considerable importance is developed, as a result, seemingly, of the very low pressure in the vessels near the fistula. Possibly the oxygen want established in the peripheral tissues hastens the peripheral vasodilatation. In any case, the collateral channels, if established in the region of a growing epiphyseal line, sometimes occasion overgrowth of a long bone. Moreover, if the fistula, together with its afferent and efferent vessels, is excised, the collateral circulation can be relied upon to nourish the extremity. However, if the afferent artery *alone* is divided, the collateral circulation carries all its blood into the fistula (easy retrograde pathways), and peripheral gangrene results.

greater in circumference than the right Its upper part was hot to the touch The hands were alike Great veins could be seen on the ventral surface of the whole forearm, but there was an especially prominent mass of them just below the back of the elbow There was felt, over the region of the elbow and forearm, a systolic thrill, and a continuous bruit was audible through the stethoscope Thrill and bruit extended along the course of the brachial vessels into the axilla, ending at a point above the clavicle The radial pulses were alike and seemed of similar force Firm pressure on the lower brachial, just above or in the antecubital fossa, obliterated both bruit and thrill Pressure over the great mass of pulsating veins upon the dorsal surface of the upper forearm nearly but not quite accomplished the same thing

The following special observations were made by Dr C S Burwell and his associates of the Medical Staff of the Peter Bent Brigham Hospital

1 Pulse rate with fistula open 84

Pulse rate with fistula closed, 71

2 Arterial Pressure	Right Arm	Right Leg	Left Leg
Fistula open	102/57	123/83	128/76
Fistula closed	117/78	142/100	142/87

3 Venous Pressure*	Right Arm	Left Elbow	Dorsal Aspect of Left Fore arm
(in mm of water)	65 mm	145 mm	265 mm

4 Circulation Time	Right Arm Vein to Tongue	Left Arm Vein (just above antecubital space)
	22.5 seconds	12.5 seconds

5 Oxygen Content of Venous Blood in Volumes per cent (Oxygen capacity 18.2)	Right Arm	Left Arm
	14.5	16.17 in forearm 14.3 at wrist

\* Elevation of the venous pressure in this case at least is apparently a local one It is most marked in the vicinity of the fistula and diminishes progressively as it is examined nearer the heart However at every point studied between the fistula and the heart the venous pressure was higher than in other parts of the body



## The Congenital Arteriovenous Fistula Case Report

It has already been explained that there is no valid distinction between arteriovenous fistula and aneurysm, but there is a rather consistent difference between congenital and traumatic communications. When in the course of the differentiation of artery from vein in the common capillary bed of the embryo, connections are left uniting the two sorts of vessels, these are apt to be multiple, to occur even over a considerable distance. Especially upon a limb, there may be a whole series of small connections between the principal artery, or one of its larger divisions, and its companion vein. A traumatic fistula, on the other hand, unites the paired vessels at some one spot, in the axilla, at the elbow, the groin, the popliteal space, or some intermediate point. And because a considerable amount of blood escapes into the tissues where the bullet or sliver passed through the two vessels, the resulting hematoma is apt to become organized and to be hollowed out into a false aneurysm connecting with both artery and vein and finally lined with endothelium. Thus, most congenital communications resemble fistulas while the traumatic ones are either direct openings or arteriovenous aneurysms. In such a situation as the neck, the two sorts of lesions resemble each other, but in a limb the congenital lesion is apt to present a characteristic appearance. The following case report gives an account of most of the features of a congenital multiple fistula in an extremity and of some of the difficulties met in treating a condition of this sort. (See Plates XII and XIII, A and B.)

J J M, a husky nineteen year old youth, had noticed nothing remarkable about his left arm until, at the age of ten, he fell out of a swing. Then the arm began to swell but never lengthened disproportionately. Dilated veins appeared about the elbow and upper forearm, especially upon its dorsal surface and after a time were seen to pulsate. The arm caused no pain or disability but because of the pulsation and palpable thrill the boy was induced to seek treatment.

Examination showed the left forearm to be seven cm

exposed. The brachial was larger than before and seemed very friable. Several small connections from artery to vein were found just above the bifurcation. Upon dividing these, the ulnar and the ulnar recurrent arteries were seen to have large connections with veins, but since distal division of these arteries and veins could not be performed from the antecubital side and since it was evident that distally placed fistulas would continue to open up as the proximal ones were obliterated, the wound was closed and further surgery was for the moment abandoned.

During the next two weeks the dilated brachial artery ruptured twice (others have noticed this accident) and was finally divided, together with the vein, half way from elbow to shoulder. The median nerve was accidentally severed and was reunited by suture.

The result, January, 1938, shows considerable improvement. The pulsation, thrill, and bruit in the forearm are gone. The hand is well nourished. However, the bruit can still be heard in the axilla. The median nerve has shown a reasonably complete regeneration.

This case illustrates the multiple character of the malformation, the dilatation and degeneration of the afferent artery, and the opening up of secondary peripheral connections as the proximal ones are closed. More especially it shows how remarkably efficient is the collateral circulation created in the presence of such a fistula. Ordinarily, division of the efferent artery results in gangrene of the limb (the blood carried by the collateral vessels flows back into the fistula). Here the proximal vein, as well, was divided (raising the pressure on the venous side and forcing blood to follow the collaterals) and successive division of several proximal fistulas probably helped to increase the resistance of the fistulous pathway and direct the stream into the peripheral parts.

#### TRAUMATIC ARTERIOVENOUS ANEURYSM AND FISTULA

In order to make clear the distinction between the various lesions which may result when an artery is pierced or rup-

- 6 Total blood volume 6770 c cm or 102.6 (c cm) per kilo (Blue dye method of Gibson and Evans) "which is well above normal even for a very muscular active man and is at a level seen in congestive heart failure"
- 7 The heart, by seven foot plate, was within the normal limits of size

*Operation, June 4th, 1936* Under ether anesthesia and with compression of the upper arm by an Esmarch bandage, an incision was made exposing the brachial vessels in the lower arm and through the antecubital space. In the upper part of the wound, for a distance of five to six cm, the brachial artery appeared twice its normal size. It then became quite rapidly smaller, reaching its natural caliber some two to three cm above its division into radial and ulnar branches. Almost four to five cm proximal to the point where the brachial narrowed, was an S shaped little vessel two to three mm in diameter and about one and a half cm in length which passed from artery to vein. This was divided and tied with silk. Then the operator, not at first realizing that this little vessel might be an important fistula, asked to have the Esmarch bandage loosened and when the current was let in, the venous pulsation, thrill, and bruit in the region of the elbow were found to have ceased!

However, an axillary thrill and bruit persisted and since these in turn were blotted out by compressing the brachial artery and vein in the wound, some connection distal to the division of the brachial at the elbow was judged to have persisted. This idea was confirmed on the following day when the expansile pulsation, bruit, and thrill in the forearm returned.

Two months later an arteriogram was made. This showed that "the opaque material enters the region of the aneurysm by means of the dorsal and volar interosseous arteries and the ulnar recurrent artery, the radial artery does not appear to communicate except through the radial recurrent"

*Second Operation, October 7th, 1936*—The former scar was excised and the lower brachial artery and its bifurcation again

point and controlling its blood flow with a rubber-covered clamp or rubber tubing, or, if this is impracticable, by making digital pressure upon it, the clot and blood can often be evacuated from the region of injury and the artery either repaired by suture or ligated above and below the opening. Naturally, the decision whether or not to attack the vessel directly or to allow a hematoma to develop to its fullest extent is a difficult one. Provided means of transfusion are at hand, direct attack is usually the method of choice.

A pulsating hematoma which has actually come to resemble a purely arterial aneurysm will present the signs of aneurysm. That is, it will have an expansile pulsation and a *systolic* bruit and thrill, in contrast to the arteriovenous aneurysm or fistula in which these signs, though accented in systole are continued through diastole. The treatment of such a false aneurysm is that of aneurysm (*q v*).

### Varieties of Arteriovenous Aneurysm and Fistula

It would be impossible to describe all or indeed many of the appearances presented by traumatic fistulas in various regions. Naturally most of the victims will be aware that they have been shot or stabbed, though a fine, flying sliver of glass or steel may perhaps fail to be noticed. Some bleeding from the injured artery is inevitable. External bleeding may occur or a large hematoma may form. However, a fistula is sometimes established so quietly that a continuous audible bruit and palpable thrill, appearing a few days later, will be the first signs of the seriousness of the injury. Occasionally, especially in wounds of the limbs, the aneurysm or fistula will make pressure on a near by nerve, causing numbness or muscular paralysis.

The rapidity with which cardiac damage may take place after the first signs of fistula have been noticed, is vouched for by a case reported by Mason, a brief account of which is abstracted from his report.

A colored woman, aged thirty, was first seen half an hour after being stabbed below the left clavicle, close to the sternum

tured it is necessary to consider several possible occurrences (1) the artery alone is injured, in which case a *Pulsating Hematoma*, or *False Aneurysm* may develop, (2) the artery and vein are injured in such a way that, following the organization of a hematoma, a connection is established between them by way of a sac, which becomes lined with endothelium—an *Arteriovenous Aneurysm*, or (3) the artery and vein are so injured that a direct connection is made between them—an *Arteriovenous Fistula*. For purposes of study and treatment, it has already been stated the arteriovenous aneurysm and fistula are alike. However, the pulsating hematoma is different and requires a separate description.

**Pulsating Hematoma, or False Aneurysm**—This is a very rare lesion and the reason for this is easy to see. When a large artery is pierced, its companion vein is usually injured as well, so that if the individual survives and if the vessels are not at once ligated or repaired, an arteriovenous connection is made. Suppose, however, that an artery alone is injured. The opening is made beneath a heavy layer of fascia. If it is not so directly connected with the outside world that the individual bleeds to death, a hematoma is rapidly formed beneath the aponeurosis. Bleeding continues until the pressure within the hematoma equals the arterial pressure. If the hematoma is then left to itself, it will tend to become so tense as to block the blood supply to the distal portion of the limb (in the case of the femoral, popliteal, axillary, or brachial, for instance) and gangrene will ensue. This is all the more likely to happen because the rapid loss of blood will have caused a decided fall of blood pressure. However, should the limb survive, the mass of extravasated blood will soon become organized at the periphery, its center remaining liquid. Only through this very rare combination of circumstances can a pulsating hematoma form, but actually it is seldom given an opportunity. For if an individual has been stabbed or shot in such a way that a large artery appears to have been injured and if a hematoma is rapidly enlarging, most surgeons will endeavor to ligate the vessel. By approaching it at a proximal

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ard treatment for arteriovenous fistula Matas has successfully restored the continuity of the subclavian by closing the rent in the artery through the opened vein, but most surgeons feel more sure of a cure in making the quadruple division and excising the fistula-connected vessels Mason gives to everyone who contemplates attacking a subclavian arteriovenous fistula the advice to begin by reading Halsted's essay on "Ligation of the Left Subclavian Artery in its first Portion" The difficulty of access is such that one should always resect the clavicle and even the adjacent upper and lateral portion of the manubrium sterni The clavicle is missed less than one would suppose and if resected subperiosteally will often regenerate

Subclavian fistulas bring up most of the difficulties inherent in treating all traumatic arteriovenous communications If the operation is performed too soon, a collateral circulation may not have become established. If performed too late, the heart will have received permanent damage or the patient will have deteriorated beyond aid If performed early, the vessels will have barely recovered from the injury and will be difficult to handle If performed late, they will have established so many unexpected connections that quadrilateral ligation will fail to still the current in the region of the fistula This last difficulty was demonstrated to the writer by Elkin in an operation upon a subclavian fistula of sixteen years' duration Incidentally, this case, by contrast with Mason's, though it had caused the heart to dilate, had led to no cardiac decompensation After three hours' work and when every visible connection with the great dilated vessels which constituted the arteriovenous aneurysm had been divided, arterial blood still passed abundantly through the fistula Any further approach turned out to be so very bloody that recourse was had to infolding the remaining vessels with a series of heavy stitches Reid has faced this situation in a similar way, and on another occasion, when all the vessels connected with the fistula could not be severed, has twisted the divided proximal vein until it was not only obliterated but completely closed the opening

To summarize the subject of the subclavian fistula it may



Her heart was known, by previous examination, to have been normal and at first presented nothing unusual. On the second succeeding day, a thrill was first noticed in the supraclavicular region, and a bruit could be heard locally and in the neck. Thrill and bruit, which were continuous, increased in violence and range during the two following days.

Sixteen days after the injury, the signs persisted, but the heart appeared undamaged. The same was true after thirty-three days.

About forty-five days after the injury, the patient was discovered confined to bed by circulatory embarrassment and at the end of nearly ten weeks was in a serious condition. The heart was enormously dilated, a pleural effusion had formed on the left, the feet and legs were edematous, the liver enlarged, the abdomen distended and dropsical. Dyspnea and cough were present. For all this, there was as yet no external swelling over the fistula.

Two operations, under a local anesthetic, were required before the fistula could be isolated by quadruple ligation and excised, so that it was over three months after the wound had been received before the strain of the great fistulous leak from the subclavian artery could be taken off the heart. As a result, the heart regained its normal size and all the other signs of decompensation cleared up, but an aortic thrill and bruit remained as well as a double mitral murmur. Mason noted the occurrence of Branham's sign when he drew tight the ligature on the subclavian artery, that is, the pulse rate fell at once (from 124 to 104). Had this difficult operation not been so skillfully and bloodlessly performed the patient would soon have died.

**Subclavian Fistula** —The case just quoted will serve as an introduction to this subject. It has been duplicated by others, though never more dramatically. There is a general agreement that division of the artery and vein proximally and distally, that is, quadruple ligation, completely isolating the fistula, is compatible with the life of the limb supplied by the injured great vessels. This may then be regarded as the stand-

tive The radial pulse was not altered All vessels were found to have opened into a common sac

Here the early operation was forced upon the attendant surgeons, yet, perhaps because the young patient's vessels were so elastic, quadruple ligation and excision were followed by no disabilities

It is hardly necessary to describe other arteriovenous fistulas of the upper limb Such a lesion may even occur on a finger Elkan illustrates one of this sort The treatment of such conditions is exactly that of the larger vessels

**Femoral Fistula**—In general, the fistulas of the lower extremity are less well borne and more prone to cause cardiac dilatation and decompensation than those of the upper Those of the femoral, because of its size, are particularly serious The limb is apt to become edematous and cyanotic, and sores upon the lower leg may develop Though the lesion is easily accessible, the many vessels centering on the region of the groin make it especially complicated The inferior epigastric and deep circumflex iliac artery and vein, the great saphenous vein, the profunda femoris artery and vein, all join the superficial femoral almost at one spot Thus a fistula near the inguinal ligament may be impossible of excision Even after dividing the proximal and distal vessels, the fistula connected artery and vein will perhaps continue to exhibit the familiar thrill It may then be possible only to transfix and plicate the actual region of the false passage with many stitches As in treating fistulas of other great vessels, it is usually necessary, as Reid tells, to ligate with tape, rather than even the largest and heaviest silk Halsted's metal bands may be useful on occasion, especially when the proximal artery is dilated and friable

**Popliteal Fistula**—This, like the true arterial aneurysm, is especially apt to interfere seriously with the nutrition of the foot Edema and cyanosis will usually be considerable A fistula in the lower femoral or upper popliteal region is easier to deal with than one in the lower angle of the popliteal space For here the popliteal artery, having given off the anterior

come on quietly or with a hematoma, it may lead rapidly to cardiac decompensation or be well borne by the patient, it may and usually does create a permanent mass of pulsating veins above the clavicle. At the risk of some cardiac damage, it had better not be attacked surgically for several months, to permit the establishment of a collateral circulation. It should be approached by an ample incision and by removal of the clavicle (and perhaps a portion of the sternum). It should be isolated and if possible excised after quadrilateral division.

**Fistulas of the Upper Limb**—The same general principles as have been discussed for the management of fistulas nearer the heart apply to these, but the indications for treatment may be somewhat different. By the kindness of Reid and McGuire, the writer is permitted to reproduce their illustrations of a brachial fistula, successfully treated by a very early operation.

The patient, No. 81335, Cincinnati General Hospital, a boy, sixteen years of age, had been struck in the right arm, just above the elbow, by a fragment of exploding pistol shell. Free external bleeding was followed by marked swelling which subsided after three days. At this time, a pulsating tumor appeared, and the usual continuous thrill and bruit were noted. There also developed a weakness in gripping the hand and a tingling sensation in the hand (pressure upon the median nerve). The tumor rapidly and painfully increased in size.

By the twenty third day after the accident, the median nerve palsy had become so pronounced, the causalgia so serious, and the aneurysmal covering so thin, that a surgical operation was imperative. The circulation time in the right arm proximal to the fistula was 140 seconds and compared to 213 seconds in the left. The heart was normal by every test. Blood pressure was 120/60 in both arms, a suggestively low diastolic pressure. On occluding the aneurysm, the pulse rate did not change but the diastolic pressure rose thirty mm. Hg.

Operation disclosed the characteristic picture shown in Plate XIII. The afferent vessels had not yet become dilated, and excision after quadruple division (or rather sextuple, since the two venae comites were involved) was cura

of blood in circulation may require the withdrawal of some of it after the fistula has been excised.

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tibial, divides into the peroneal and posterior tibial branches. It is easy, then, to understand that when these are connected with the fistula or with the aneurysmal sac, division of all afferent and efferent vessels is almost impossible. It is not as if one could control the arterial supply merely by compressing the main vessel proximal to the fistula. Cut off this vessel, and the distal branches at once pour in a retrograde current. It may thus be necessary, after all accessible arteries and veins have been ligated, to plicate the remains of the sac, or the vessels joined by the fistula, with heavy silk.

#### COMMENT ON THE TREATMENT OF TRAUMATIC ARTERIOVENOUS ANEURYSM AND FISTULA

In planning and executing a surgical operation, the following considerations must be kept in mind

- 1 The collateral circulation must have time to develop, but if more than a few months are allowed to pass, the local connections of the fistula may become so numerous and complicated that quadruple ligation is insufficient to effect a cure. Then, special means may be required to obliterate the fistula and its connecting vessels.

- 2 If treatment is delayed, serious damage may be done to the heart, which, therefore, must be very carefully watched.

- 3 If the heart is so damaged that it can not be expected to withstand a radical operation, division of the proximal vein will lighten its load and perhaps permit it to recover.

- 4 Ample exposure is absolutely essential.

- 5 In exposing the fistula, the afferent artery should be reached and isolated as soon as possible and should then be controlled by the use of a piece of soft rubber tubing.

- 6 The afferent artery should never alone be divided. If division of the afferent vessel seems imperative, the afferent vein or veins should also be ligated.

- 7 The afferent artery is often exceedingly friable and requires great care in ligation. It may subsequently undergo aneurysmal dilatation or actually rupture.

- 8 Since loss of blood at the operating table may be excessive, a sterile system of suction should be used to conduct blood from the wound into a receiving bottle containing sodium citrate. It can then readily be infused into a vein while the operation proceeds.

- 9 If very little blood has been lost during the operation, the excess

## CHAPTER VIII

### LYMPHANGIOMA ELEPHANTIASIS LYMPHEDEMA

THE lymphatic system is an independent anatomical unit, having the function of absorbing all such tissue fluids as the blood capillaries are unable to accept and, in addition, foreign particulate matter. These fluids, filtered through intervening groups of lymph nodes, it forwards to the superior vena cava. In many ways, it resembles the venous system, with which it makes only two connections. Of these, the principal one is at the point where the left jugular vein joins the subclavian. Here the thoracic duct, carrying lymph from the lower limbs and lower half of the trunk, chyle from the lacteals, in fact, the lymphatic drainage from all of the body except the right upper quadrant, enters by a broad delta. The small, short duct on the right, receiving only the lymph from the right arm, right pectoral region, and right side of the neck and head, enters the right subclavian vein at a corresponding point. The lymph vessels, down to those of the very smallest size, are furnished at frequent intervals with valves, so that lymph is forced through them by muscular pressure from without, much as is the case with the veins. Indeed, without some movement, lymph tends to accumulate in the limbs and its flow is decidedly quickened by active exercise.

The ultimate lymph spaces, in the form of an epithelial lined network, permeate the skin and subcutaneous tissues everywhere, a closed system of capillaries soon gathered together into somewhat larger but still hardly visible vessels which take very much the same course as the veins. The long lymphatics appear to run in several planes, (1) a very superficial one, in the true skin, where they are easily seen when infected and inflamed, as in the case of acute tubular lym

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18 REID, MONT R "Studies on Abnormal Arteriovenous Communications, Acquired and Congenital", *Arch Surg*, 10 601, March, 1906, May, 1925 11 25, July, 237, Aug, 1925

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21 THEIS, F V "Popliteal Aneurysms as a Cause of Peripheral Circulatory Disease with a Special Study of Oscillomographs as an Aid to Diagnosis", *Surgery*, 2 327, Sept, 1937

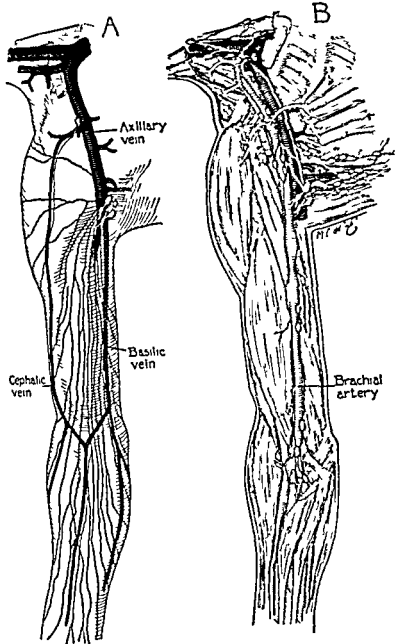


FIGURE 26 THE LYMPHATICS AND NODES OF THE ARM AND AXILLA The arm is abducted to a right angle with the body—may be viewed from the long side of the page. *A* The superficial lymphatics and their relation to the veins and axillary nodes. Note the assemblage of nearly all of the superficial lymphatics at one point in the lower axilla. *B* The deep lymphatics and their relation to the principal artery. The superficial lymphatics join them in the lower axilla. The course of the lymphatics follows Rouvière's descriptions.



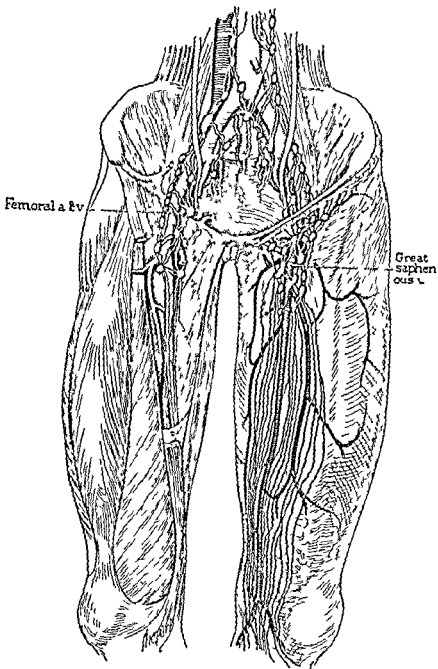


FIGURE 2. THE LYMPHATICS OF THE LOWER LIMBS In the left leg, the superficial lymphatics and nodes are shown, especially their relation to the superficial veins In the right leg, the deep lymphatics are associated with the principal arteries The lymphatics of the pelvic brim are drawn from a dissection by the late Dr John Warren of the Harvard Medical School (Courtesy of *Annals of Surgery* 100 S15, Oct 1934, and the *New England Journal of Medicine*, 204 1025, May 14, 1931 )

dant and few other than the serious tropical infestations of various sorts are able to bring on an elephantiasis by an obstruction at the root of a leg or arm.

Much is now known of the anatomy of the lymphatics but as to one important matter no really authoritative statement is forthcoming. They do not appear to drain the muscles. \* Lymph vessels running in the intermuscular connective tissue spaces are recognizable but the circulation in muscle itself seems to be carried on by blood vessels and blood capillaries only. That this is actually so is strongly suggested by the absence of any detectable lymphedema in muscle when the superficial tissues of a limb are grossly distended with fluid and crippled by the fibrosis of elephantiasis. For though the limb is then heavy and unwieldy, the muscles remain normal and function naturally. Nor has it ever been shown, in spite of many attempts, that the accumulated fluids of the surface can be introduced among the muscles and so drained from a limb.

**Embryological Considerations**—The tree like organization of the lymphatics in the extremities is most obvious if they are pictured as budding from a single point at the root of each limb, as Sabin has shown. When this development goes wrong, a malformation of the superficial tissue may occur, giving rise to puffy, spongy thickenings or actual cyst formation. Such malformations, or lymphangiomas, may occupy a part or all of a lip or tongue or cheek or hand or foot, may follow the distribution of several dermatomes, or may even cover a whole limb. In many of these lesions, dilated lymph vessels are evident, in others, irregular spaces without any great collections of tissue fluids are found. By contrast, congenital cystic malformations of the lymphatics do occur, especially at the root of the neck or of a limb, huge multilocular cavities difficult to deal with by surgical means.

**Physiological Considerations**—The fluid which bathes the cells of the tissues is derived from blood, that is, from the

Bouvière states (page 4) that 'The muscular lymph networks consist of very fine capillaries which are arranged in a variegated manner around the muscle fibers.' He quotes Agard (1913) in support of this statement but to the writer Agard's demonstration is not convincing.

phangitis, and (2) a deeper one corresponding to that of the superficial veins, that is, in the subcutaneous fat or actually lying upon the aponeurosis. The accompanying illustration shows how groups of these vessels follow, in the leg, the course of the principal branches of the great saphenous vein and, coming together at the saphenous opening, join the trunks which course along the femoral artery and vein. This third and last group consists of only two or three rather larger but still fine vessels which drain the foot, the back and outer side of the leg (through the local lymphatics which empty into the popliteal space) and probably the intermuscular connective tissue. A very similar condition obtains in the upper extremity. Most of the superficial lymphatics enter the mid axilla in order to pass into the axillary glands of this region, while the deep trunks, which are associated with the various great blood vessels, drain the palm and receive in the lower arm the lymphatics which have already passed through the epitrochlear group of glands. An occasional lymph vessel passes into the upper axilla in association with the cephalic vein or even jumps the clavicle.

In both the upper and lower limbs, each great collecting tree has a very narrow base, at the groin and axilla respectively, after which a group of large lymphatics, about a millimeter in diameter and varying in size and number, winds about the principal blood vessels, the artery in particular, to join the thoracic duct. There is, then, a long bottle neck between groin or axilla, as the case may be, and the great collecting duct which is to pour the lymph into the superior vena cava. At such a bottle neck the drainage of any one limb can, theoretically, be rather easily interrupted, especially where the lymphatics pass through the great groups of regional lymph nodes. As a practical matter, however, the flow is not often cut off at these points, save by operations performed for cancer of the breast, that is, infections, considering the frequency with which they cause an extensive axillary or inguinal adenitis, rarely lead to lymphedema of a limb. Apparently alternative routes and emergency passages are abun-

It is hard to imagine any injection of foreign material which does not rupture many lymphatics, but in any case motion is evidently a prime factor in the lymphatic absorption of foreign material, for without it the process is exceedingly slow.

Leaving out of consideration the generalized edemas due to an altered chemistry of the blood, most accumulations of tissue fluids in the legs are of a mechanical nature. That is, the lymphatics are absolutely deficient or (on the functional side) more tissue fluid is present than a normal absorbing system can accommodate or, the fluid and absorbing system being normal, the elastic pressure of muscular action is lacking. There will therefore be included in this chapter congenital malformations, elephantiasis, edemas resulting from thrombophlebitis, edemas related to allergy and to infection, and functional edemas related to injury and disuse.

### LYMPHANGIOMAS, CONGENITAL MALFORMATIONS OF THE LYMPHATICS

As is the case with the hemangiomas and abnormal arteriovenous communications, the actual growths of lymphatic tissue and the various sorts of congenital malformations so merge into one another that a clear distinction between them is impossible. Almost all subcutaneous swellings of lymph vessels are present at birth, and grow with the body, though they may enlarge disproportionately—particularly the cystic ones. If a localized lymphangiomatous or lymphangiectatic swelling, not present at birth, grows in after years, it is likely to be mistaken for a lipoma or plexiform neuroma. Such an event is very rare.

Malformations of the lymphatics are much less common than the corresponding disorders of the blood vessels. They have been divided into three classes, *simple*, *cavernous* and *cystic*, and for the sake of clearness the various sorts will be briefly described here, whether or not they are likely to be found upon the extremities.

**Simple Lymphangioma (Lymphangioma Simplex)**—T

arterial side of the capillary bed. A part of it, consisting principally of water and salts, re enters the blood capillaries, but plasma proteins and all foreign materials are carried off by the lymphatics, whose task it evidently is to remove from the vicinity of the body cells substances whose presence in abnor

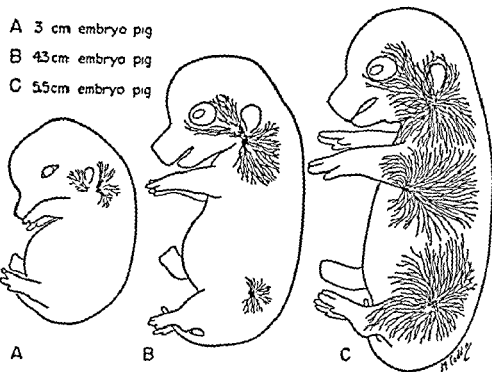


FIGURE 27 THE DEVELOPMENT OF THE LYMPHATICS IN THE PIG Showing how they grow out upon the limbs Modified from sketches of Dr Florence R Sabin

mal amounts would harm the organism. Obviously the high percentage of colloidal material in the blood capillaries opposes the entrance into them of extravascular proteins, whereas the lymph capillaries, whose function is purely absorptive, are quite able to receive them. Just how foreign bodies, such as red cells, carbon particles, and other dusts, enter the lymphatics is not understood. Of course they can be, and are, carried in by phagocytes, but they are undoubtedly taken up mechanically as well, though whether they are forced through the capillary wall, or infolded by it, or pushed in, after actual rupture of the delicate endothelial cell membrane, is unknown.

oped and afterwards changes little in size, except as it grows with the infant.

Many lymphangiomatous deformities include malformations of the blood vessels, so that it is only possible to say that something has gone wrong with the development of the whole superficial vascular system in the region affected. The surface of an entire limb may be malformed, as a result of which bizarre changes occur. Groups of nipple-like projections from which fluid is discharged at intervals have been described. The writer has encountered a case of this sort in a young girl. At the time of her menstrual period a brownish fluid would be discharged from these little excrescences, apparently because of some change brought on by the menstrual cycle, a sort of vicarious menstruation. In other cases, the projections appear to consist of local dilatations extruded above the surface, capable of discharging a great deal of fluid, if injured. The boundaries of these cavernous lymphangiomas are vague. They merge at their edges into normal tissues. The back of several fingers may be covered with a soft spongy thickening, which fades out upon the back of the hand. The scar like, lymph soaked, vascular tissue of the growth has no clean-cut deep surface but is directly attached to the underlying muscle, tendon, or whatever, without the intervention of the usual aponeurosis. It may even grow into intermuscular planes in the form of wedge shaped processes.

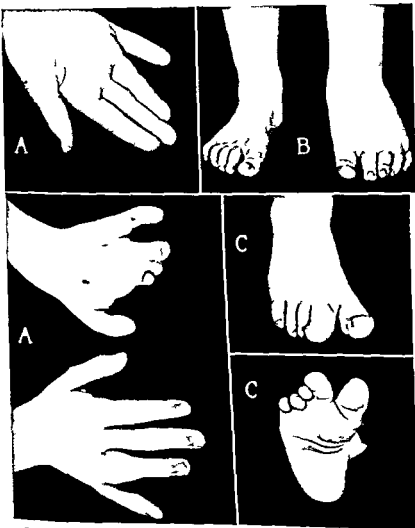
*Treatment* of these lesions is very difficult. Excision is the ideal method but anything like a complete, clean-cut removal is rarely possible. Considerable masses can, however, be excised in a series of operations, by turning back flaps of skin, excising the exposed lymphangiomatous tissue down to normal muscle, bone, or whatever lies beneath, and replacing the skin flaps upon these sound parts. In such operations the blood supply is rather difficult to control and the patient is exposed to the risk of infection. However, by not attempting too much at any one time, much may be done. As an alternative, the tissues may be punctured over many sittings with the electrothermy needle and caused to shrink considerably.

sort is confined to the skin and subcutaneous tissues. It takes the form of a soft, ill-defined swelling, easily compressible, over which the skin is unchanged. Occasionally, very superficial, large, clear lymph vessels can be seen through a very thin cutaneous covering. Simple lymphangiomas occasionally appear upon the hands and feet.

The writer's experience includes none of these simple lymphangiomas of the extremities but instances are reported of bands of lymphangiomatous tissue passing out upon a limb and containing good sized vessels. These vessels, if injured, are capable of causing a long continued leak of lymph, or lymphorrhœa.

The treatment of such angiomas is surgical excision, or, in case this is impracticable, the use of the endothermy needle. Possibly some of them are more or less radiosensitive.

**Cavernous Lymphangioma (Lymphangioma Cavernosum)**  
—This lesion, which merges into the simple sort, a malformation or growth according to your fancy, is a spongy mass of dilated lymph spaces lined with endothelium. It appears as a smooth swelling which may cause great enlargement of such a part as the tongue (macroglossia) or the lips (macrocheilia). On a surface like that of the fingers or the back of the hand it forms a puffy thickening upon which the natural folds of the part may be lost. The skin over it is usually normal in color, and the connection with entering vessels is free enough to permit the surface of the mass to be indented. However, after many years, especially upon the lower limbs, the tissues may become so fibrosed that pitting is only produced with difficulty. With these swellings bizarre deformities of the fingers and toes are often associated, as is shown in one of the accompanying Plates. The digits may be of giant size or stumpy or webbed or missing. However, the deformed toes or fingers are not necessarily themselves the seat of lymphangioma. They may merely exhibit fibrosis and edema. A distinction between lymphangioma and a congenital elephantiac lymphedema is not always clear, but as a rule the elephantiac part progressively swells, whereas the lymphangioma appears fully devel-



**LYMPHANGIOMAS IN CHILDREN** *A* R F A, 132043 There is a diffuse swelling of the base of the ring finger and ulnar side of the right hand. A partial excision has already been performed. The left hand is malformed. (Courtesy of Dr. T. H. Lanman, Children's Hospital, Boston.) *B* J B A, 180144 Age 12 months. A diffuse swelling of the left foot, dorsal surface. *C* G A 86022 Age 22 months. A malformation partly lymphangiomatous, of the right second toe. (Courtesy of Dr. Augustus Thorndike, Children's Hospital.)



**Cystic Lymphangioma (Lymphangioma Cysticum) Cystic Hygroma**—This form, actually a benign growth, is composed of huge multilocular lymph spaces and is seen more often in females than males. Its favorite seat is the root of the neck, but it may appear nearer the jaws, in the axilla, or even, very

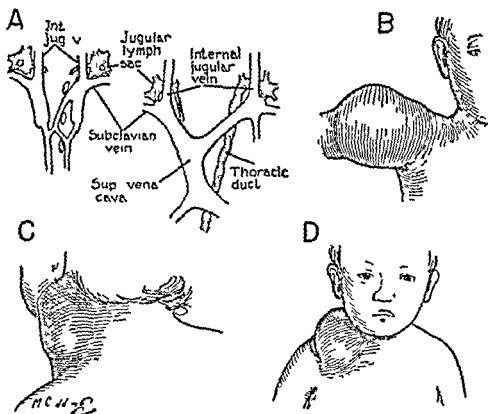
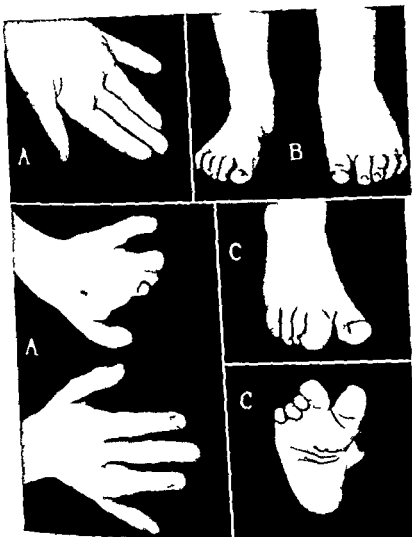


FIGURE 28. **HYGROMA** A The primitive jugular lymph sacs (shaded) of the human embryo and the formation of the thoracic duct (Kampmeier) B, C, and D The cystic hygroma in various situations. Drawn from representative illustrations in the publications of Dowd and of Goetsch.

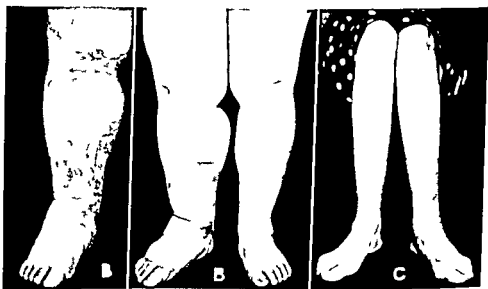
rarely, in the groin. It is really a maldevelopment of one of the primitive lymph sacs, especially one of the jugular sacs, which fails to become united with the rest of the lymphatic system. Its peculiar manner of growth has been explained by Goetsch, who finds, budding from its surface, endothelial sprouts from which microscopic fibrils force their way into adjoining muscle, causing atrophy and fatty degeneration of the fibers they isolate. Moreover, the pressure exerted by the



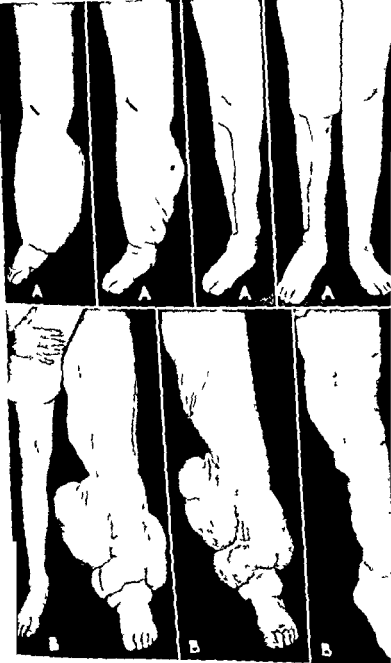
LYMPHANGOMAS IN CHILDREN. I REF A 18-13 Thorndike Children's Hospital. II  
 in, of the base of the ring finger and ulnar side of the little finger. A partial  
 excision has already been performed. The left hand is malformed (Courtesy  
 of Dr T. H. Lanman, Children's Hospital, Boston). D J B A., 18-131 Ag 1  
 two A diffuse swelling of the left foot, plantar surface. C G A C 18-131 Ag 1  
 two A malformation partly lymphangomatous of the right foot to  
 (Courtesy of Dr Augustus Thorndike, Children's Hospital)



1 A VERY EXTENSIVE LYMPHO-HEMANGIOMA RNM, 43115, a girl, aged fifteen Notice the capillary hemangiomatous patches and the peculiar spot, which discharged a bloody fluid with the exudate, upon the larger swelling at the groin and on the back of the lower thigh



B IP, 51493 aged 36 Infectious type of Elephantiasis In the central figure, note ulcers on the right On the extreme left this same leg (dark red) during an inflammatory attack Treated by lumbar sympathectomy Since then no more attacks and most of ulcers have healed C TH, 20609, (ODD), aged 56 Bilateral edema of an allergic sort Repeated attacks of allergic edema due to fungus



FLEPHANTIASIS NOSTRA A HSL 0461 A woman aged thirty  
 From left to right before elevation after elevation for a week after  
 onl stage after fourth stage B HFW 5629 a man aged forty  
 From left to right after elevation for several days during an attack  
 in man r dness), after several operations The scars on the thigh  
 result of old lion toleon proce lures



growing cyst causes it to envelop structures in its path. Yet the hygroma is never malignant.

Being a growth of congenital origin, the hygroma usually appears, more often just above the right clavicle than elsewhere, in the first year of life, and before two years have passed, has become an orange-sized swelling, fluctuant, perhaps translucent, but not to be obliterated by pressure. The accompanying sketches, made after the Figures of Goetsch and of Dowd, will give an idea of the nature of the tumor. Only when it first shows itself, as it occasionally does, in childhood or adolescence rather than infancy, is it likely to be confused with such lesions as branchial cleft cysts, soft or cystic neurofibromas, angiomas, hematomas, or broken down lymph nodes. The fluid contents of the hygroma are thin, clear, watery, and contains as a rule a low percentage of protein, so that it does not coagulate on exposure to the air, and since it is independent of the lymphatic vessels in general, it does not cause swelling of the limb at whose root it happens to be situated. It may, however, force its way into the mediastinum, enveloping the great vessels in its path.

*Treatment* is excision. Radiation or the injection of sclerosing solutions can not be recommended. Operations, for the most part, are performed in the second year of life. Some are very difficult, especially when the hygroma invades the mediastinum. It is important, if the operation is to leave nothing behind, to excise the entire mass unruptured.

**Lymphadenocoele**—This name is given to a swelling of lymphatic origin, particularly at the groin, which appears to be derived from cystic enlargement of the regional lymph nodes. Such a swelling may perhaps be related to cystic hygroma or to filarial infections. In the latter case, the female filarial organisms may be found in the lesion. Any cystic disease of the lymph nodes at the root of a limb is of course likely to be associated with a lymphedema of the whole limb, that is, elephantiasis, a subject next to be considered. It is difficult to see how a lymphadenocoele can be treated by any method other

than excision and why excision should benefit (or aggravate) obstruction of the lymphatics of the limb involved

### ELEPHANTIASIS

There are certainly three and probably four varieties of elephantiasis, if one chooses to accept a classification based on the manner in which the disease makes its appearance. However, since the means by which lymphatic drainage is put out of action is far from clear even in tropical (filarial) elephantiasis and is utterly unknown in the sporadic and hereditary sorts, classification had better be as simple as possible. Accordingly, the varieties presently to be described are distinguished from each other sometimes on pathological and sometimes on clinical grounds without much thought of scientific accuracy.

1 **Surgical Elephantiasis** (*Elephantiasis Chirurgica*), usually results from operations performed for mammary cancer but very occasionally for serious protracted infections or malignant tumors of the lymph nodes themselves. Thus, most instances of this sort occur in the arms.

Elephantiasis of the arm, following a radical operation for mammary cancer, appears to be related to destruction of the great collection of superficial lymphatics as they enter the axilla to join the deep perivascular lymph vessels. It happens in a freakish way, most often perhaps when the regional nodes are already infiltrated with cancer but on occasions as well when cancer is altogether absent, as after operations performed under a mistaken diagnosis. Nor does it seem necessary, as Halsted assumed, that infection should be a factor. That the lymphatic obstruction is not due primarily to interruption of the perivascular lymph vessels coming up from the lower arm is suggested by the anatomical fact that those lymphatics are related to the artery (and even the great nerves) rather than to the axillary vein. Actually, of course, it is the vein rather than the artery which is exposed and cleared of lymphatic tissue in the radical operation upon the breast.

It is the writer's experience that elephantiasis of the arm

is most likely to follow a radical mastectomy (extensive cancerous invasion of lymph nodes not being a factor) when a considerable accumulation of lymph has appeared in the operative field during the week or ten days following operation, that is, when there is evidence that a considerable body of lymphatics has been interrupted. Upon cessation of the leak, swelling of the arm is apt to begin. However, swelling may rarely appear, disappear, and recur, as if lymph drainage were teetering on the edge of success for some weeks or even months. Once a surgical lymphedema sets in, it is almost always progressive, for when too much of a task is put on a few remaining vessels, there is experimental ground \* for believing that they may easily dilate and so become functionless. Moreover, postoperative elephantiasis of the arm shows itself first in the forearm rather than in the hand, whose lymph drainage seems to persist for some time via the deep lymphatics after that of the superficial tissues of the arm and forearm has been interrupted. Recently, Veal has analyzed various types of elephantiasis of the arm and has found that there is a pure lymphatic variety, a sort due to venous obstruction and a mixed type. He regards recurrent cancer as mainly responsible.

A surgical elephantiasis of the arm behaves, in respect to the peculiar febrile attacks so familiar in permanent lymph stasis, about like any other elephantiasis, and is subject, unpredictably, to the onset of these attacks. But because the arm is less dependent than the leg, and more easily drained of its tissue fluid by gravity, its elephantiasis is usually rather less disabling. Only when recurrent cancer aggravates the obstruction by actually blocking veins and tissue spaces as well as lymphatics is surgical treatment likely to be demanded. Then, of course, amputation is almost the only recourse. Should relief be desired in less serious cases, the operation advised for elephantiasis of the leg may be used (page 310). It should be remembered that even though the lymphatics are destroyed,

The writer has observed dilatation and finally complete loss of function in nearly obstructed lymphatics in the dog. See reference at the end of the chapter.



fluids still flow by gravity through tissue spaces, but the tissues have now lost the advantage of the valved vessel in forwarding fluid against gravity. Fluid, therefore, flows out of the arm on elevation and accumulates in it on dependency. Elevation plus exercise drains it most effectively.

With surgical elephantiasis should be included the terminal lymphedemas of the leg associated with malignant growths in the female pelvis. These are clearly due to cancerous invasion of the lymph vessels and nodes along the pelvic brim, as by ovarian or uterine cancer. There should also be included the serious edemas which may ensue when a mass of primarily malignant lymph nodes is treated by surgical excision plus irradiation.

**2 Elephantiasis due to Infection**—The serious progressive lymphedemas which arise from this cause are apt to be associated with some open lesion such as an ulcer. Through this, the infectious organisms reach the lymphatics and presumably interrupt them by obliterating the regional lymph nodes. It is almost always the leg which is affected. In some cases, it is difficult to decide which is the cart and which the horse. Does the lymphedema arise from repeated infection, or does the elephantiasis occasion, at an unusually early stage, the attacks of cellulitis so characteristic of the advanced disease and so hasten its own development? The leg, in cases of this sort, is apt to be enlarged and tense at all times, having little tendency to the drainage and wrinkling on elevation which marks most other elephantiasis. Moreover, there is a suspicion of an allergic reaction in some cases, a hypersensitive ness to fungi or bacteria entering through the lesions of epidermophytosis such as will be described in a later section. The elephantiasis based on infection is rare in temperate regions and becomes increasingly common, as Matas has stated, when tropical climates, favoring infection of all sorts, are approached. Under such circumstances, a femoral thrombophlebitis occasionally leads to a disease of this kind. Unless open ulcer or evidence of infection clearly forbids plastic operations, such treatment as is used for the commoner sorts

elephantiasis is suitable for this form. Actually, preventive treatment is the main thing. Fungus and bacterial infection must be fought off by appropriate means. In this respect the writer has more than a suspicion that the vasodilatation secured by lumbar sympathectomy may prove useful.

3 **Elephantiasis Tropica**, the filarial sort, is due to permeation of the lymphatics of the limbs by the parasite, especially the large female form. Except in those bizarre elephantiasis in which a lymphadenocoele is present, and except that the arms are sometimes involved and that the secondary febrile attacks are more often observed in tropical countries than elsewhere, there is nothing about this form of the disease essentially different from the hereditary and sporadic varieties. The manner in which the lymphatics are destroyed is different but the end result is much the same. Considerable numbers of calcified worms can be found in the tissues, as O'Connor, Golden and Auchincloss have demonstrated by the aid of the X ray. These may also be present in individuals who show no sign of elephantiasis. Tissues containing them have been excised by Auchincloss in an effort to remove tender spots and areas which have persistently ached, areas which have seemed to the patients to be starting points for their inflammatory attacks. His removal of such elephantiac tissues led him to advise operation for this purpose only and not with the idea of securing drainage of the lymphedema as such. Since this general plan of surgical treatment now seems to offer the best chance of success in the nontropical as well as the filarial sorts of elephantiasis, it is presented below.

4 **Elephantiasis Nostra**. Milroy's and Meige's Disease — These are lymphedemas exactly similar both pathologically and clinically, but for which no cause whatever can be discovered. Milroy and Meige described independently an hereditary elephantiasis as each saw it in a family. Hope and French have written a most picturesque account of another family. Almost all of Milroy's cases were not only familial but congenital, that is, the elephantiasis developed from birth, whereas in the other families the condition first showed itself,

as a rule, in the neighborhood of puberty. The disease exhibits all the characteristics of sporadic elephantiasis, its situation, progressive course, tendency to inflammatory attacks and to gross terminal deformities. It therefore requires no separate description.

Elephantiasis nostra, or sporadic elephantiasis, is not particularly uncommon. Certainly it is vastly more often seen than the familial form. More would be heard from it were it not that most of its victims are not only ashamed to show their legs but are told that nothing can be done for them. Both sexes are affected, females perhaps the more often. The onset is usually at about the time of puberty, rarely at birth or during childhood, occasionally after the twentieth year and very rarely indeed after thirty. As a rule, the disease is confined to one leg, but perhaps one case in eight or ten is bilateral.

As in filarial elephantiasis, enlargement of the leg is gradual. The ankle is swollen first, then edema mounts to the knee and finally invades the thigh. In the beginning, the skin is unchanged and the superficial parts pit on pressure, but as time goes on, the skin becomes thick and the subcutaneous tissue hard. After some years, pitting no longer takes place unless the parts are made to soften and wrinkle by elevation of the leg for several days. By the yielding of the skin here and there sacculations of strange shape are made. These are apt to be separated by deep creases. All changes are less well developed in the thigh than the lower leg and the swelling stops cleanly at the inguinal ligament and the crease below the buttock. If a shoe is constantly worn, the foot does not enlarge and the appearance of the skin which it covers does not change. That is to say, continuous pressure, in the form of a boot or anklet, keeps the tissues drained of fluid. The blood supply of the elephantiac leg, so far as can be judged by the color of the skin and the surface temperature, is entirely normal, and so it certainly appears at the operating table.

The explanation of these clinical findings is to be found in the state of the tissues and tissue fluids both in the experi

mental animal,\* in which the disease has almost exactly been reproduced, and in the human patient. As the tissue fluids accumulate, their content of protein increases until it may reach a height of even four per cent—half that of blood serum. With this change, fibrosis advances, perhaps because the highly proteinized fluid acts as a tissue-culture medium. Characteristic coarse trabeculations become visible in soft tissue roentgenograms and constitute, as Reichert has shown, a feature of the disease. All such reactions seem to be proportional to the degree of stasis. It has already been explained that they are almost unnoticeable in the area of foot covered by the shoe and that they are far less marked in the thigh, from which fluid can rather easily escape into the abdominal wall or retroperitoneal tissue, than elsewhere.

Efforts to demonstrate a valved lymphatic, or indeed anything like a normal lymph vessel, in the elephantiac leg have been fruitless. There may be great spaces containing fluid either just beneath the skin or upon the thickened aponeurosis. But the injection of trypan blue, which the lymphatics readily take up and retain, fails to demonstrate any paths for tissue drainage except such spaces. Sometimes the dye injected into the deep skin will run out rapidly, making a diffuse splotchy stain. Indeed, if the leg is elevated, it may pass up from the foot to the thigh in a few minutes, just as is the case in the elephantiac leg of the dog, though in other instances, it merely makes a local blue mark and moves hardly at all. If, however, a patient elevates the leg at an angle of thirty degrees for a few days, the fluid may reach the body so rapidly as to form a swelling in the corresponding flank and skyrocket the urinary output. As a rule, if the leg has not been elevated, puncture with a needle anywhere in the calf will obtain a flow of clear lymph, but sometimes the engorged spaces are not reached by the needle, so that hardly a drop of fluid will flow.

The deep as well as the superficial lymphatics are totally absent. The writer has explored the femoral vessels of the

\* Drinker, Field and Homans. Also Homans, Drinker and Field. See Bibliography at the end of the chapter.

as a rule, in the neighborhood of puberty. The disease exhibits all the characteristics of sporadic elephantiasis, its situation, progressive course, tendency to inflammatory attacks and to gross terminal deformities. It therefore requires no separate description.

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The explanation of these clinical findings is to be found in the state of the tissues and tissue fluids both in the experi-

strual periods or in connection with a respiratory infection, especially, perhaps, when the patient is tired or run down. In a recent case treated by the writer, the young man, following the usual plastic operation, remained free from attacks (which had previously made an invalid of him) for some two years. Then, during a very damp hot summer, an old epidermophy tosis of the foot recurred and he suffered a febrile attack. Probably streptococci entered by way of the fungus infection. It has recently been shown that in the experimental elephantiasis of the dog, the attacks, like those of man, may set in spontaneously. This has offered the opportunity, which Drinker and Field seized, to secure tissue fluid just as the attack reached its height. They found in the fluid, at this moment, with perfect regularity, a limited number of streptococci. The same strain would persist in the attacks for many months, and then mysteriously a new strain would appear. The local heat, swelling, fever, and prostration exactly imitated the human disease. However, soon after the attack had reached its height and between attacks, no bacteria could be found in the tissues. Doubtless in man the attacks are of similar character, and in parts of the world where the disease is common have been treated by polyvalent streptococcal sera with considerable success. Possibly sulfanilamide will prove effective against them and should undoubtedly be tried on the ground that the streptococci are probably hemolytic.

Advanced elephantiasis, associated with extraordinary malformations, frequent febrile attacks and perhaps local ulcerations, is a very serious disease. A case in point is illustrated herewith, that of a man whose attacks had worn him down so that not only did his unwieldy leg keep him from work and all enjoyment of life but he had become dull and depressed, a chronic invalid. Removal of much of the diseased tissue has made a new man of him. Even if his attacks should occasionally recur he will have taken on a new lease of life. It has been shown furthermore that in such a case as his, bacteria are present in the tissues at all times and that operations must be

elephantiasis leg without finding any lymphatics about them. However, on the occasion of a pelvic exploration, in the case of a young woman suffering from elephantiasis of both legs and the external genitalia, the great perivascular iliac lymph vessels were actually observed. They were large, by reason of being thick walled, as if chronically inflamed, and actually contained the least bit of lymph, so that some remnant of lymph flow remained. However, it could not be determined what tissues they were draining (probably those of the pelvis only) and the lymph nodes were curiously flattened and atrophied. Whether such an appearance is the rule is unknown.

*The Febrile Attacks* of elephantiasis are dramatic and peculiar. They rarely appear until the disease is well advanced. Many persons are free from them altogether. Indeed, in both the sporadic and hereditary varieties of the disease, which are alike also in other respects, perhaps not more than one patient in four suffers from this complication. However, once attacks are established, they continue at long intervals or short as the case may be. In a typical instance, the elephantiasis leg first feels uneasy and within a few hours becomes hot, red, and additionally swollen. The temperature rises so rapidly that a chill, often a very severe one, is almost always felt. The infection is distinguished from others by involving the whole limb at one time, not in a creeping manner. The temperature rises to  $102^{\circ}$ – $104^{\circ}$  F ( $39^{\circ}$ – $40^{\circ}$  C) and the patient is often severely prostrated. But for all that, the attack is self limited. It usually lasts for three to five days, being totally uninfluenced by treatment.

The cause of the attack, which is far more common in the filarial elephantiasis of tropical countries than in the elephantiasis nostra of temperate climes, has always seemed obscure. It is usually dubbed erysipelas or cellulitis, yet bacteria have seldom been recovered from the tissues. Moreover, the timing of the attacks has always been erratic. They may occur about once a month or once in six months, with men

lar spaces beneath the aponeurosis To that end, long incisions were made, flaps were turned back, lymph soaked tissue and aponeurosis were removed and the skin flaps were replaced upon the muscles It was thought also that pathways could be made to lead fluid to the body by prolonging the incisions from the leg over the hip to the flank or over the inguinal ligament in front These latter schemes were based on Handley's plan of inlaid silk strands which were intended to drain the elephantiac arm after radical mastectomy Unfortunately there is no such thing as restoring lymph drainage, nor does a scar conduct fluid to the body as effectively as nature's widened subcutaneous tissue spaces Thus any benefit which ensues upon plastic operations seems to be due to the removal of those tissues in which the fluid is formed and retained The ideal procedure is, then, the making of the thinnest possible flaps of skin, removal of all soft parts down to healthy, muscle, tendon and bone—none of which seem to accumulate tissue fluid—and replacement of the skin flaps upon these deep structures Sistrunk went a good way toward accomplishing this, Auchincloss frankly planned to do away with (filarial) elephantiac tissue and the operation described below seems to go about as far in the way of reducing the amount of subcutaneous tissue as it is possible to go Moreover, since the lower leg is the chief reservoir of fluid and is subject to the greatest deformity, there is no reason in most cases for carrying the plastic higher than the knee Actually the thigh is reduced in size by the complete operation below, for less fluid now has to pass through it on the way to the body, and the thigh itself is rather readily drained by elevation.

The accompanying sketches will clarify the following brief description. Under a general anesthetic, the leg, foot, and toes are thoroughly cleansed by any routine method Then, when everything is ready to procede, an Esmarch bandage is applied in a broad band to the thigh. This may have to be reinforced by rubber tubing, for the elephantiac thigh is compressed with difficulty and the operative field must be bloodless

An incision is then made from just below the knee to the



performed with great care. No sort of incision, much less an elaborate plastic, can be made in his case without exciting infection and sloughing (Plate XVII B).

*Non-Operative Treatment*—On the ground that fluid can escape from the elephantiac leg only by way of tissue spaces, it would seem that unless the individual spends his life with his feet higher than his head, his leg must gradually enlarge. Such is not quite true or at least not true in all cases. The wearing of a firm anklet and bandage will keep some legs fairly free from accumulated fluid and the shoe is so effective that the same size can usually be worn on the foot of the elephantiac leg as on the other. If the individual, in addition to receiving the aid of this sort of pressure, is able, when sitting, to elevate the leg upon a stool or chair, for a good part of the day, and does not have to stand for long hours, drainage of the leg is still further favored. Finally, the foot of the bed should be raised, if possible, six inches, so that gravity may help to empty the limb at night. The writer knows of a working girl who, having invented these means for herself, has used them so successfully as to be able to control the swelling of her leg. Possibly her tissue spaces are particularly adapted to such treatment, but she can actually pass an evening in dancing provided she is willing to use a little extra elevation before and after. Of course, a leak of lymph, such as may follow any little injury, keeps swelling down, but the excessive wetness is not exactly compatible with normal activities.

Fungus infections must be overcome. They offer an entrance to bacteria and in that way, as already explained, may occasion the streptococcal attacks. Ulcerations, if present, are a similar menace. However, in any but the very advanced case they are not apt to occur, for, in temperate regions at least, most elephantiac tissues heal about like any others.

*Operative Treatment*—There has been a good deal of misunderstanding about the objects of the sort of plastic operation, based on Kondoleon's original procedure, which has usually been performed. Kondoleon's idea was to drain fluid from the lymph soaked superficial tissues into the intermuscu-

in at the row of little perforating arteries and veins given off by the anterior tibial vessels, lateral to the tibia. Then the incision is carried down through the muscular aponeurosis so that the flaps, when turned up, lay bare periosteum, muscle, tendon sheath and even the capsule of the ankle joint. Superficial nerves are as a rule ignored but the posterior tibial vessels and nerve are carefully avoided.

The great flaps are turned back so as to expose more than half the circumference of the leg. Then the thin skin flaps are defined. For perhaps two thirds of the width of each, the flap is formed of bare skin, the deep skin being cleaned just as if a whole thickness graft were being made. However, at the base of each flap some fat is left, particularly where draining veins are visible, for the difficulty with these flaps is that they have too little venous drainage, not too little arterial supply. The flaps on the foot are made less completely of skin than those of the calf. Just how much of the edge of each flap to remove is a problem. Much extra skin is of course present, and it would seem that the final tension upon each flap must resemble that of the skin in its original state (just as is the case with a full thickness skin graft).

Once the skin flaps are made, the fluid soaked tissues beneath are removed, the base of the great masses being divided with heavy scissors. At this point, the Esmarch bandage is released from the thigh and all bleeding points are picked up with fine snaps and ligated with fine silk, the object being to leave the least amount of injured tissue possible. The field must be left particularly dry if the flaps are to heal ideally. The best results are secured by tacking them down with rows of fine silk stitches, one line in the middle of each flap and the third uniting and holding down their edges, so that very few skin sutures are needed. It will be well to score the flaps lightly near their edges with a sharp knife, which drains their venous blood, just before the dressing is applied and pressure is made. A large loose gauze dressing should be fitted into the broad trough left by the plastic, held in place with a firm muslin or semi elastic bandage and supported by a light plas-

foot, having at the top a broad symmetrical Y and at the bottom an inverted Y of which the forward arm is much longer than the other. Thus lower inverted Y is placed below the internal malleolus. The incision is made upon the anterointernal face of the leg so that the base of the lateral flap will have

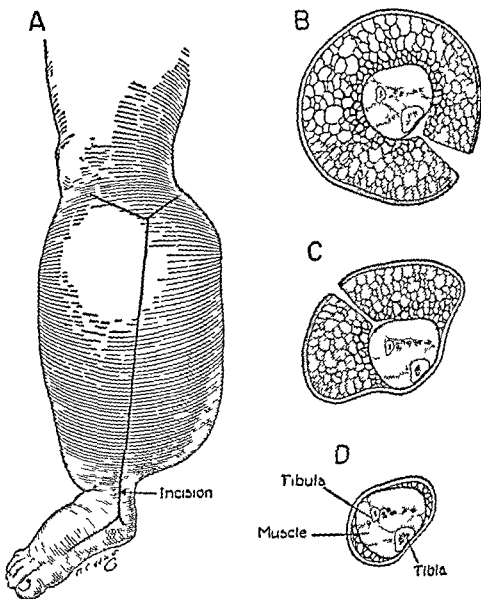


FIGURE 29 THE TWO STAGE OPERATION FOR ELEPHANTIASIS A The first plastic (antero medial) B Cross section of leg showing what is to be removed (shaded tissues) at the first stage C End of first stage and plan for second D End of second plastic (Postero lateral incision)

be supposed that the lymphatics are not seriously diseased or perhaps that only some particular groups are affected. One can easily conceive that the sort of perivascular inflammation described in Chapter VI as so often engulfing the principal vessels of the limb, may destroy or cripple some of the lymph trunks which wind about the femoral and iliac artery and vein. On the other hand, a moderate venous stasis might keep the tissues soaked in a fluid which only the lymphatics could carry away (high protein content). This might be just too much of a task for them and edema would naturally result. Until more is known of the vasospasms and venous valvular deficiencies which follow thrombophlebitis, an authoritative explanation of this sort of edema can not be offered. However, the occasional appearance, after a femoro iliac thrombophlebitis, of signs of vascular spasm or even a reflex dystrophy of the extremity, suggests that a vasomotor disorder may be venous and persistent.

*Treatment*—Some regard postphlebotic edemas as purely of venous origin others, as lymphatic. Actually, for purposes of treatment, the distinction is not very important. The problem is to permit the individual the greatest amount of activity with the least possible resultant swelling. First, because a swollen limb is a nuisance and second, because continued edema encourages fibrosis and the postphlebotic indurations and ulcers already described. Compression by the semi-elastic cotton bandage or the elastic stocking is very valuable, but the individual, like the sufferer from elephantiasis, must plan his or her life for a due proportion of periods of elevation of the limb. And of course any varicose veins which may have developed from collateral venous enlargements during the period of actual obstruction may require surgical treatment. Here the recognition of incompetent communicating veins is very important. Injection is usually contraindicated, and painless taking excisions of the veins is, as a rule, demanded. A description of the diagnostic and technical procedures required for the treatment of such varicose veins will be found in Chapter V. In occasional instances, when evidence of vascular

ter cast Thus support is necessary both to prevent foot drop and unexpected strains on the flaps

Originally, the writer advocated a four stage procedure, treating one-fourth of the leg's circumference at each stage but the two stage procedure saves much time and seems to result in no serious sloughs Even the necrosis sometimes shown by the skin in patches at the edges of the flaps is apparently shallow However, the operation can doubtless be improved upon by the thoughtful surgeon

The second stage is performed after an interval of perhaps two months, when it may be expected that new flaps can safely be made with their bases toward the old ones The incision now is postero external and since a part of the aponeurosis upon the lateral surface of the leg is inseparable from the underlying muscle, a little of the actual tendinous muscular sheath is necessarily cut away The sural nerve is saved if possible, since it supplies the lateral side of the heel and foot Otherwise the operation is similar to the first one Possibly the whole procedure ought to be divided into three stages instead of two, for then the three sets of perforating arteries, and roughly corresponding veins, could be preserved—the antero lateral (anterior tibial), antero mesial (posterior tibial) and postero lateral (peroneal) A supporting bandage is worn during the first few weeks following the final procedure The patient must of course continue to avoid long hours of standing and elevate the leg whenever possible

**Lymphedema following Thrombophlebitis**—The local edemas, indurations and ulcerations which follow phlegmasia alba dolens (femoro iliac thrombophlebitis) were described in Chapter V, so that they could be compared with varicose ulcers The sort of edema referred to here is that which, in occasional instances, follows this same form of thrombophlebitis and may rarely persist throughout life It is easily distinguished by the history and by the fact that it affects the entire limb It differs from elephantiac lymphedema in that it is not progressive but stationary, increasing by day and nearly or entirely disappearing at night It must therefore

after weeks or months in bed, or if muscles or joints are so injured that normal muscular contractions and motions are not permitted, fluid accumulates. Lymph flow, like the flow of venous blood, as already explained, greatly depends on motion and is rather readily disturbed.

Much of this sort of disability can be avoided or at least lessened by exercises and massage during the period of a serious illness and by an arrangement of apparatus during the treatment of sprains and fractures such that the function of muscle and joint can be preserved. Nevertheless the sprained ankle, unless skillfully treated by adhesive strapping and active exercise, may result in many weeks of swelling.

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spasm or reflex dystrophy is present, an interruption of the reflex pathway by periarterial or lumbar sympathectomy is indicated

**Lymphedema resulting from Allergy and Infection**—It is difficult to segregate the lymphedemas of this sort they must be considered very similar to the swellings of the lips associated with chronic infections of the sinuses, which Stevens described some years ago and with which those dealing with allergic manifestations are doubtless familiar As a result of repeated attacks of infection or allergic swelling, a leg or legs may gradually enlarge, becoming progressively indurated but not ulcerated Such a state, resulting in elephantiasis, has already been described The writer has the impression that the fungus infections may be responsible for some of these swollen legs and the streptococcal infections which enter by way of the lesions of epidermophytosis for others It is very difficult to separate actually infectious from allergic attacks and the matter is all the more confusing in that the protein of dead fungi or of dead bacteria may equally well constitute the exciting cause in any one case

*Treatment* of such an indurated swelling includes a thorough study of sources of infection and of the possibilities of hypersensitiveness to fungi and bacteria Desensitization may then produce good results Surgical treatment, save for the possible usefulness of a permanent sympathetic vasodilatation in overcoming a chronic cutaneous infection, has little to offer

**Lymphedema related to Injury and Disuse**—Such must be described as functional and for the most part temporary They should not be confused with the "traumatic edemas" related to the dystrophies and cruralgias described in Chapter IV These latter are arterial in nature, whereas the sort described here are due to a failure of muscular effort to aid the lymphatics in the disposal of tissue fluids

The borderline between swelling and no swelling is a narrow and delicate one The circulation of the lower extremities has had to adapt itself to the upright position which imposes an unnatural burden upon it If, therefore, the muscles atrophy,

after weeks or months in bed, or if muscles or joints are so injured that normal muscular contractions and motions are not permitted, fluid accumulates. Lymph flow, like the flow of venous blood, as already explained, greatly depends on motion and is rather readily disturbed.

Much of this sort of disability can be avoided or at least lessened by exercises and massage during the period of a serious illness and by an arrangement of apparatus during the treatment of sprains and fractures such that the function of muscle and joint can be preserved. Nevertheless the sprained ankle, unless skillfully treated by adhesive strapping and active exercise, may result in many weeks of swelling.

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## CHAPTER IX

### INTERPRETATION OF SOME SIMPLE OBSERVATIONS UPON THE CIRCULATORY DISORDERS OF THE LIMBS

IN THE opening chapter of this book, the various circulatory disorders were named and roughly sorted out. Methods were described by which, once their nature was suspected, they could be studied, how, for instance, an arterial deficiency could be ascribed to arteriosclerosis, to thrombo angutis obliterans, or to peripheral arterial spasm, recurrent or chronic. No serious attempt was made, however, to discuss the various possible meanings of common symptoms. Actually, the circulatory diseases of the limbs have a rather limited number of ways of showing themselves. They cause pain or numbness, coldness or heat, cyanosis, pallor, swelling, atrophy, or gangrene, usually a combination of several of these changes. It is by the manner of onset of such symptoms and signs, their combinations and their background of age, sex, and associated disease that a diagnosis is unmistakably indicated. Widely different disorders may sometimes look much alike, arterial embolism and venous thrombosis, for instance. Even those most familiar with the meaning of outspoken, familiar signs may be confused. All the more will those who rarely meet them find certain appearances meaningless and bewildering. It is proposed, therefore, to discuss here some common presentations, complexes, showing how, by relatively simple observation and reasoning they may be explained.

#### PAIN

Pain, as a rule, means arterial deficiency. If it comes on as a result of exercise and disappears on rest, it represents

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## CHAPTER IX

### INTERPRETATION OF SOME SIMPLE OBSERVATIONS UPON THE CIRCULATORY DISORDERS OF THE LIMBS

IN THE opening chapter of this book, the various circulatory disorders were named and roughly sorted out. Methods were described by which, once their nature was suspected, they could be studied, how, for instance, an arterial deficiency could be ascribed to arteriosclerosis, to thrombo angustis obliterans, or to peripheral arterial spasm, recurrent or chronic. No serious attempt was made, however, to discuss the various possible meanings of common symptoms. Actually, the circulatory diseases of the limbs have a rather limited number of ways of showing themselves. They cause pain or numbness, coldness or heat, cyanosis, pallor, swelling, atrophy, or gangrene, usually a combination of several of these changes. It is by the manner of onset of such symptoms and signs, their combinations and their background of age, sex, and associated disease that a diagnosis is unmistakably indicated. Widely different disorders may sometimes look much alike, arterial embolism and venous thrombosis, for instance. Even those most familiar with the meaning of outspoken, familiar signs may be confused. All the more will those who rarely meet them find certain appearances meaningless and bewildering. It is proposed, therefore, to discuss here some common presenting complexes, showing how, by relatively simple observation and reasoning they may be explained.

#### PAIN

Pain, as a rule, means arterial deficiency. If it comes on as a result of exercise and disappears on rest, it represents

*intermittent limp*, and indicates thrombo anguitis obliterans or arteriosclerosis. In either case, it may be referred to the calf or shin or foot and will vary in severity from numbness, through cramp, to a sharp stab. It is thought to be due to an accumulation of certain metabolites in the tissues.

Pain which comes out of a clear sky, an agonizing persistent pain, usually means sudden arterial closure. Several causes of such closure should come to mind, depending upon the circumstances under which it appears. With all sudden arterial closures, absence of the peripheral pulses, coldness, local paralysis, marbled bluish pallor, and very often some slight degree of swelling are likely to be associated.

1 Arterial thrombosis (Chapters II and III) The patient will be one who, because of advanced age, is subject to arteriosclerosis or whose earlier age, sex (male) and story of intermittent limp are consistent with thrombo anguitis obliterans.

2 Arterial embolism (Chapter IV, last part) The patient, who may be of any age from youth on, will exhibit a fibrillating heart, evidence of mitral stenosis or coronary disease, or of acute cardiac decompensation. Immediately premonitory numbness or coldness will often have occurred.

3 Venous thrombosis, in the femoral or external iliac vein (Chapter VI) Usually an operation, or injury, or debilitating disease, or childbirth, will be a background for the thrombosis. This form, phlegmasia alba dolens, is often ushered in by severe pain, referred to the inner face of the thigh, the back of the knee or the calf, an indication of ischemia due to reflex arterial spasm. The peripheral pulses may be weak or absent, the leg cold and white. Such a state is apt to be temporary—being followed in a day or so by the characteristic swelling—and only rarely goes on to the full state of coldness, mottled pallor, and final gangrene, with no or little edema, characteristic of the organic occlusions.

Arterial thrombosis, arterial embolism, and arterial spasm secondary to venous thrombosis need not necessarily cause pain. Coldness, numbness, and paralysis may be the initial

signs The gangrene which so often follows, tends to be of the moist type

Pain of a persistent continuous sort, especially in the toes and forefoot, associated with cyanosis and a shiny swelling, together with some degree of gangrene of one or more toes, indicates a serious arterial deficiency and threatens loss of toes or even foot and leg The most severe pains are due to thrombo angitis obliterans, but arteriosclerotic cyanosis and gangrene sometimes cause great suffering The pain is usually made worse by elevation and is diminished, temporarily at least, by moderate depression of the foot The diabetic gangrenes seem to cause little pain (Chapters II and III)

### CYANOSIS, PALLOR, AND COLDNESS

Cyanosis, pallor, and coldness will be discussed together because they are due to a much retarded and usually restricted circulation Pallor represents constricted vessels, cyanosis, a slow current, irrespective of whether the cause lies in the arteries or veins (Chapter I)

In the fingers (rather than the toes) coming on acutely and temporarily in response to cold or emotional upsets, these signs are due to Raynaud's Phenomenon, that is, spasm of the digital arteries (Chapter IV)

The sudden establishment of cyanosis, pallor, and coldness in any limb, whether or not preceded by severe pain or merely by numbness and paralysis, indicates sudden arterial occlusion, which, as already explained, may be embolic, thrombotic, or spasmodic (associated with thrombophlebitis) If the coldness persists, if the pulses below the root of the limb disappear, if the mottling becomes a fixed purple, and if these changes affect the lower half of the limb, such changes are irreversible and gangrene will follow Swelling is variable, but the blue, cold area is at least full and often blistered

*Explanation* That some degree of cyanosis and even edema is usually associated with sudden arterial stoppage need not cause surprise The veins of the terminal parts may even be

prominent When the arterial stream is slowly cut down until it entirely ceases to supply the foot, for instance, the leg gradually atrophies and is, so to speak, drying up even before the final stage of gangrene On the other hand, when blood flow is rapidly cut off, the tissues are caught wet Blood accumulates in the finest blood vessels, having nothing to push it along, and losing its oxygen, while the leg is still warm, gives the skin a deep purple color Very likely the oxygen want due to the sudden arterial stoppage causes the fine vessels to dilate, so that they accumulate the last of the blood delivered to them Actually there seems sometimes to have been an escape of blood into the subcutaneous tissues, as in post mortem lividity, for often the purple color of the mottlings or large patches can not be altered by pressure But even if pressure causes the purple skin to become pale, such a change should not be interpreted as proving that a circulation exists and that the tissues are alive The stagnant blood will flow back into the pale spot with a speed proportional to its accumulation in the neighborhood Only if the limb is elevated, can the manner in which a spot, blanched by pressure, regains its color, be interpreted as meaning anything at all If, while the foot is *elevated*, the blanched spot regains some degree of color, then the circulation is still going on and the part is alive (For a further interpretation of color see Chapter I)

The occasional absence of pain, when a large vessel is suddenly occluded, is very puzzling Any leg which rapidly becomes numb, powerless, more or less edematous, blistered, and pulseless, which takes on within twelve hours a mottled, bluish whiteness, and which, at the end of thirty six hours, appears partly white, partly livid, and utterly cold, has certainly suffered a sudden shutting off of its arterial stream That this can happen without pain is certain, yet the ischemia of an embolic arterial occlusion, when painful, is as agonizing as anything encountered in medicine Morphine may have no appreciable effect upon it

**Chronic Cyanosis and Coldness of the Hands and Feet** are rather common *In young people*, usually but not always fe-

males, this indicates continuous arteriolar spasm. Hands and feet are equally affected. Excessive sweating of the cyanotic parts is almost the rule. Obviously the state is therefore one of continuous sympathetic irritability, since the parts affected are those most responsive to sympathetic changes. Warm surroundings lessen but never abolish the cyanosis, they merely change the tint toward red or even pink. Exposure to cold and emotional strains intensify the sympathetic irritability. The peripheral arteries present normal pulses. In the presence of such a state, sympathetic paralysis, however produced, turns the skin warm and pink, that is, organic changes in the finer arteries have not occurred, and the condition is always curable by sympathectomy.

Chronic cyanosis and coldness in *elderly people* usually means arteriosclerotic deficiency. Peripheral pulses may still be present but more often are absent.

### THE PERIPHERAL ARTERIAL PULSATIONS

The Dorsalis Pedis artery is best palpated upon the forward part of the instep, just lateral to the first metatarsal bone, and two to four fingers' breadth proximal to the (distal) head of that bone. This artery is, as a rule, more easily felt than the posterior tibial, and its pulsations are used as the standard for the state of the peripheral vessels of the leg. It almost never happens that, under normal conditions, both dorsalis pedis arteries are insignificant or absent, but if such is the case, the posterior tibials will be particularly strong.

Absence or enfeeblement of one dorsalis pedis pulse is suggestive evidence of arterial deficiency, and in that case the posterior tibial will, naturally, be equally deficient. Such is true of thrombo angustis obliterans and all acute arterial obstructions. Absence of the dorsalis pedis is not, however, necessary to a diagnosis of arteriosclerotic or of diabetic arterial deficiency or gangrene. In fact, arteriosclerotic gangrene of a toe occurs fairly often in the presence of a pulse, though usually a weak one, in the dorsalis pedis. This is even more likely to happen in a diabetic, in whom an arterial de-



prominent When the arterial stream is slowly cut down until it entirely ceases to supply the foot, for instance, the leg gradually atrophies and is, so to speak, drying up even before the final stage of gangrene On the other hand, when blood flow is rapidly cut off, the tissues are caught wet Blood accumulates in the finest blood vessels, having nothing to push it along, and losing its oxygen, while the leg is still warm, gives the skin a deep purple color Very likely the oxygen want due to the sudden arterial stoppage causes the fine vessels to dilate, so that they accumulate the last of the blood delivered to them Actually there seems sometimes to have been an escape of blood into the subcutaneous tissues, as in post-mortem lividity, for often the purple color of the mottlings or large patches can not be altered by pressure But even if pressure causes the purple skin to become pale, such a change should not be interpreted as proving that a circulation exists and that the tissues are alive The stagnant blood will flow back into the pale spot with a speed proportional to its accumulation in the neighborhood Only if the limb is elevated, can the manner in which a spot, blanched by pressure, regains its color, be interpreted as meaning anything at all If, while the foot is *elevated*, the blanched spot regains some degree of color, then the circulation is still going on and the part is alive (For a further interpretation of color see Chapter I)

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**Chronic Cyanosis and Coldness of the Hands and Feet** are rather common In young people, usually but not always fe-

portance are changes from a good pulsation to a poor one or to complete failure. A sudden deterioration is almost invariably a serious sign.

In States of Acute Coldness and Numbness, due to the arterial stupor which occasionally results from direct trauma to a large artery, as by a bullet wound or fracture (especially at the elbow), the peripheral pulses rapidly disappear and may again, though seldom so rapidly, return (Chapter IV). A very special form of spasm of a large artery, the brachial, may very rarely occur suddenly as a result of irritation of its sympathetic nerve supply where it joins the lower end of the brachial plexus in its passage over the highest rib. Usually, nervous symptoms predominate, and the arterial closure is incomplete and chronic.

The sudden disappearance of the peripheral pulses because of arterial thrombosis or embolism, or as a reflex with the onset of a femoro iliac thrombophlebitis has already been described. The ischemia of all such states is usually painful but rarely is a cause merely of coldness and numbness.

#### SWELLING OF A LIMB, WITHOUT A CHANGE OF COLOR

Leaving out of consideration cardiac edemas and those due to an altered chemistry of the blood, edema of a limb occurs because more fluid collects in the subcutaneous tissues than the drainage system can carry away. Such an edema may be caused by venous stasis, by muscular atrophy and weakness after an illness, or by loss of the function of lymphatic drainage.

When a whole leg swells from toes to groin within a period of a day or two, especially after an operation or injury, or childbirth, or in the course of a serious illness, but rarely during active life, a femoro iliac thrombosis is almost necessarily present (Chapter VI). That is, any edema which rapidly mounts to a point above the knee implies that the upper femoral and probably external iliac vein (at least) are thrombosed and obstructed. There may be tenderness over the femoral vessels. The great saphenous vein, secondarily throm-

iciency is aggravated by a lack of resistance to infection (Chapter II)

*The explanation* of this distinction between the gangrene of arteriosclerosis and thrombo angutis obliterans lies in the patchy, erratic character of the arterial narrowing and obliteration in arteriosclerosis. Nor are the vessels which furnish the collateral circulation evenly distributed and of regular caliber. By contrast, the collateral circulation of thrombo angutis obliterans, if adequate, is regular and composed of very many fine vessels. Thus a patient subject to this latter disease and lacking all peripheral pulses may, unless an especially rapid closure of a good-sized artery occurs, go about for years suffering from an intermittent limp, yet with no obvious change in the nutrition of his toes. Whereas an arterio sclerotic sometimes loses a toe or two by gangrene even though the beat in his dorsalis pedis is distinctly perceptible. Diabetics, as already explained, are apt to suffer from infection plus gangrene, infection plus necrosis of a phalanx, or infection plus destruction of a toe-joint in the presence of surprisingly good peripheral pulsations.

**Absence or Enfeeblement of the peripheral pulses in persons over fifty** is not particularly unusual. Many of these are conscious that they can not walk fast without bringing on an intermittent limp, yet they adapt themselves to this state of things and say nothing about it. Their legs are rather atrophied but the color of their toes is unchanged and their toe nails are not deformed. On the other hand, some individuals bitterly resent being obliged to walk slowly and persistently seek aid. No treatment is likely to be of any great benefit to these, though abstinence from tobacco and the use of Allen Buerger exercises often help considerably. On the other hand, they may live to a considerable age without ever losing even a toe.

It will be realized that the significance of the peripheral pulsations in the foot is not always easy to perceive. The pulses have one meaning in arteriosclerosis, and especially in diabetes, another in thrombo angutis obliterans. Of most im

and the subcutaneous tissues harden. Ulceration may occur. In the presence of any such reaction, evidence of infection with a fungus, in the form of dry desquamating patches on the toes or foot or cracks and white patches between the toes, should be sought. The fungus is not likely to be present in the thickened skin of the calf. There is not necessarily any particular discomfort.

Swelling of any one extremity following a wound or injury and associated with an oversensitive skin indicates "traumatic edema," "reflex dystrophy," or whatever the causalgia like state is to be named (Chapter IV). The cause usually seems insufficient—a blow without fracture, the bite of an animal, a punctured infected wound, a mild burn—but may be serious, as in the case of a nerve injury or postphlebotic state. The edema is slight or considerable, developing slowly. The part may be cool or warm, reddish or slightly cyanotic, the peripheral pulses diminished. The change, however, which really identifies the disease is the hypesthesia of the skin. There actually is loss or diminution in the patient's sensibility to light touch and oversensitiveness to a pin prick, scratch, or pinch. Mild degrees of this state are common, easily overlooked, and frequently recover of themselves. Serious forms are rare, progressive, and incapacitating.

Swelling of one or both legs, gradual and without evident cause, is due to elephantiasis, that is, complete failure of lymphatic drainage (Chapter VIII). The swelling is at first most noticeable at the ankle but within months or years involves the whole leg, to the inguinal ligament in front, to the fold of the buttocks behind. As a rule, there is no suggestion of preliminary infection or of exposure to any tropical disease. The swelling usually begins in the 'teens. At first it is compressible but later feels very solid and does not pit on pressure. The skin is thick. The foot remains of normal size where it is compressed by the shoe. There is no limit to the swelling, which may take a bizarre form.

This gradual swelling without apparent cause separates

Increased protein and a pellicle forming in 4 to 6 hours or overnight, suggests the presence of tubercle bacilli which collect pellicle. The pellicle should be picked out and stained for acid fast organisms.

The cell count in the spinal fluid may be facilitated by drawing methylene blue staining fluid to the one mark on the red cell count and drawing the spinal fluid up to the 101 mark. Erythrocytes are almost colorless, and leucocytes will be stained blue. The average number of blood cells in one large square multiplied by ten will give the number of cells per cubic centimeter. The number of erythrocytes indicates traumatic bleeding.

The differential count is done by drawing off the supernatant, transferring the sediment to an albumin coated slide, drying, and counting by Wright's blood method.

### Myelography

Pantopaque may be obtained from the Eastman Kodak Company and is recommended for myelography particularly in the diagnosis of ruptured intervertebral discs. Its low viscosity facilitates injection and examination of the subarachnoid space, as well as easy removal by aspiration and absorption.

It is administered by subarachnoid injection as for ordinary lumbar puncture.

Lipiodol may also be used in the diagnosis of intradural tumors. It is prepared as Lipiodol 10 % Iodine Radiologique Ascendant and is instilled just as described for Pantopaque. It is produced by E. F. Johnson and Co., New York City.

Lipiodine is put out by the Ciba Company of Summit, New Jersey, for radiopaque studies just as described for Lipiodol or Pantopaque.

## PART IV

# THERAPEUTIC METHODS

### Chapter XXVI

## EMERGENCY DIAGNOSTIC PROCEDURE IN COMATOSE OR UNCONSCIOUS PATIENTS

Medical emergency situations frequently present problems of diagnosis and the physician must be prepared at all times to cope with these and save the lives of his patients. The development of coma or unconsciousness creates a serious situation which requires prompt diagnosis and treatment. The patient may suddenly or gradually lapse from semi-coma into coma exhibiting at times rapid at times slow progression. Profound or deep coma that produces an absence of supra-orbital pressure reaction and bilateral areflexia indicates a poor prognosis.

*Precursory symptoms and signs* as giddiness vertigo weakness paresthesias visual disturbance as dimness unsteadiness as staggering restlessness drowsiness confused speech somnolence to sopor, are the common complaints.

*Prominent physical signs* are pallor or red florid or cyanotic color facial palsy with drooling and a flat or blowing cheek dilated or pin point equal pupils light reflexes sluggish or brisk in both eyes. The fundi may show prominent vessels with hemorrhage or exudate or both with or without papilledema.

The skin may be hot and wet cold and wet or hot and dry, the response to stimuli may be lost the respiration may be slow deep shallow fast Cheyne Stokes or Kussmaul pulse may be weak fast or slow blood pressure may be low high or mounting convulsive movements may be present.

The situation as a whole and the surrounding circumstances should be hastily appraised. A rapid preliminary survey is made for any obvious signs of *trauma* or *abnormality* that demands immediate attention as *apnea* calling for artificial respiration *dyspnea* or *cyanosis* of cardiac tamponade that calls for oxygen clearing of airways or pericardial paracentesis *asystole* or *shock* that requires cardiovascular stimulants or free venous *oozing* or arterial spurting hemorrhage that demands immediate hemostasis.

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Unilateral flaccid palsy occurs in early hemiplegia and spastic palsy later this calls for repeated neurological examinations

Palpate scalp lacerations with sterile gloves and the underlying skull for depressions or fractures

### Eye Examination

Note pupils whether constricted or dilated, equal or unequal Pass fingers in the visual fields close to the eyes Defense closure of patient with complaint of blindness suggests hysteria Soft eyeballs suggest diabetic coma firm eyeballs occur in glaucoma Note conjugate deviation of the eyes on moving the head to the right and to the left, upward and outward suggesting hemorrhage into the ventricles

Pin point pupils suggest morphine poisoning but also hemorrhage into the ventricles or into the pons Dilatation of one or both pupils may occur in cerebral hemorrhage or neoplasm Pupils fixed to only light suggest general paralysis or tabes dorsalis especially when accompanied by the absence of deep reflex changes

Ophthalmoscopy—Examination of the ocular fundi may show choked discs or hemorrhage of the retina or exudate Papilledema occurs in hypertensive encephalopathies brain tumor abscess or subdural hematoma Retinal hemorrhage and exudate may occur in malignant hypertension eclampsia or uremia

### Facies

Blowing of one flat and flaccid cheek, drooling or retraction of the angle of the mouth indicates facial palsy Tapping the facial nerves in front of the ear reproduces Chvostek's sign of tetany in the presence of low blood calcium alkalosis or uremia In this state spasmodic twitching may occur in any part of the body

### Reflexes

To determine the muscle tone of the limbs palpate the muscles and test their strength Test the deep reflexes of the arm, leg and foot and the superficial abdominal and cremasteric reflexes Scratch the flat surface of the feet and the toe extension of Babinski is evident in a pyramidal tract lesion Unilateral flaccid palsy occurs in early hemiplegia and spastic palsy later Unilateral hyperreflexia and bilateral hypoflexia occur in meningitis Bilateral areflexia is found in profound coma Deformity of the spine or gibbus indicates fracture and calls for careful orthopedic and neurosurgical handling



A systematic physical examination should be done quickly yet thoroughly in every emergency. Note any *abnormal position* assumed as orthopnea or opisthotonus. The nature, site or source, and amount of any *bleeding* as a trickle of blood or cerebrospinal fluid from the ears or nose suggest probable fracture of the base of the skull. Frothy, pink stained phlegm is usually coughed up from a congested lung with a ruptured pulmonary blood vessel. Bloody vomitus may come from the esophageal varices, hemorrhagic gastritis, or ulcer.

Note *pallor, cyanosis, or sweating* which may be due to shock, hemorrhage, circulatory failure or cardiac tamponade, but cyanosis may be due to pneumonia, an epileptic seizure, head injury or methemoglobin. Note whether the skin is cold and clammy, wet and hot or dry and hot. Cherry red coloration of the skin is usually due to carbon monoxide poisoning.

Note any odors of *breath or vomitus*, particularly that of alcohol, acetone, phenol, ether, or chloroform.

Note *respiratory disturbances*, dyspnea or Cheyne Stokes breathing which occur in serious cardiovascular diseases and also in cerebral vascular disease and in hemorrhage, uremia, brain tumor and pneumonia. Biot's shallower and irregular incomplete apneic periods occur in meningitis.

Note hyperpnea or Kussmaul respiration which suggests diabetic acidosis and slow, deep sighing inspiration which suggests alkalosis or cholelith.

*Palpate* the neck, abdomen, or extremities for muscle rigidity of meningitis, peritonitis, spastic paralysis or flaccidity of acute central lesion type.

*Percuss* for absence of liver dullness which suggests a ruptured viscus. Hepatic atrophy and ascites suggest cholelith.

*Auscultation* of chest may reveal pneumonia, cardiac valvulitis, tachycardia or asystole. Spontaneous or convulsive movements, opisthotonus and rigid neck suggest subarachnoid hemorrhage, meningitis and tetanus.

*Convulsive syncope* occurs in Adams Stokes attacks of ventricular asystole or ventricular fibrillation from hypoglycemia, spontaneous or from insulin, hypertensive encephalopathy, uremia, paresis, brain tumor, heat and sunstroke, subdural hematoma, cholera, dysentery, hepatic disease such as acute yellow atrophy, cholelith or pancreatic apoplexy.

*Trismus or risus sardonicus* suggests tetanus. *Subsultus tendinum* is seen in meningitis and toxic encephalitis.

Decerebrate muscular rigidity occurs in encephalitis and brain injury.

There is a critical level of blood pressure below which the circulation is inadequate and tissues suffer damage. Damage to the medullary centers from prolonged low pressure probably accounts for loss of vasomotor tone late in shock.

The loss of blood volume by hemorrhage or shock raises this critical level that is the less the volume the higher the pressure required for an adequate circulation. Hence the desirability for the early restoration of blood volume and of blood pressure. This critical blood pressure level is considered to be 70 to 60 mm. Hg.

### Organic Brain Diseases

Organic brain disease epilepsy paresis or brain tumor may contribute to sudden attacks of unconsciousness or convulsions.

Epilepsy is suggested by a familial history of attacks frequent recurrences of convulsive seizures in which the tongue is bitten and the patient froths at the mouth. A history of antecedent aura and slight petit mal seizures as well as major convulsive grand mal attacks, are common in epilepsy patients.

Often the convulsion which may begin in one extremity and become generalized may be preceded by a cry. The head rotates and is drawn backward the eyes usually are deviated the pupils dilated, and the mouth drawn to one side. The hands are clenched with the thumbs often inside the fingers the fists are flexed on the wrists arms flexed at the elbows and the legs are extended. Respiration may be temporarily arrested by tonic spasm. Initially there is pallor which changes to livid as clonic spasms follow and the breathing is resumed. Sphincter control is usually lost.

If the patient is 30 or more years of age and has his first convulsive attacks one must consider malignant tumor of the brain. The complete neurological examination must be done carefully. Study the ocular fundi with an ophthalmoscope for papilledema. Electroencephalograms may be helpful in the diagnosis. Ventriculograms are usually depended upon for accurate localization.

Paresis or parietic dementia may likewise present convulsive seizures and unconsciousness and the patient may remain in coma for many days. The pupils are small irregular and fixed in paresis. Stolid smooth facies and irregular unequal Argyll Robertson pupils especially with primary optic atrophy tremulousness of the tongue and deep reflex changes may be considered characteristic of general paralysis of the insane. Blood serology and spinal fluid serology are usually positive. The cerebrospinal fluid shows an increase in lymphocytes plasma cells globulin and a typical high parietic gold curve.

### Circulation Studies

Electrocardiograms are made promptly if the pulse rate heart rate or heart action are abnormal or there has been chest pain or severe dyspnea or previous cardiovascular disease. Blood pressures, both systolic and diastolic are depressed with sino atrial tachycardia in shock hemorrhage, severe injury, adrenal insufficiency, and cholemia. The blood pressure rises progressively after severe intracranial injury and is accompanied by increasing bradycardia.

Blood pressure elevation with coma occurs in essential hypertension of the malignant cerebral type in early uremia pituitary or adrenal tumor. A blood pressure fall in a comatose patient with acceleration of the pulse and heart rate is ominous.

### Trauma to the Head

Trauma is the commonest cause of unconsciousness and must be carefully looked for in every case of coma. Trauma to the head produces changes in the brain according to the severity of the blow and the grade of brain injury which has been produced. Cerebral concussion may be mild or severe. Unconsciousness may be slight temporary or prolonged coma. Shock, sweating dilated pupils tachycardia, respiratory difficulties, and asphyxia may develop as well as other focal medullary signs. Restlessness excitement, delirium, and convulsions indicate cerebral confusion.

Bleeding into the skull may be indicated only by increased intracranial pressure. Occasional lucid intervals increase in the size of one pupil loss of reflexes slowing of the pulse all suggest the development of a hematoma which has caused increased intracranial pressure.

A trickle of blood from an ear indicates fracture of the skull laceration of the dura and possible other brain tissue damage. X ray films of the skull should always be made in all cases to determine whether a basal fracture is present and how extensive it may be. Depressed fracture of the skull should be elevated it producing irritation. When skull injury and brain laceration have been demonstrated a neurosurgeon should be called in consultation. However when there is no fracture of the skull the patient should be kept under medical observation. Usually he needs to be treated for shock intracranial pressure changes circulatory disorders, dyspnea and disturbances in organic brain function.

Post traumatic peripheral circulatory collapse occurs following uncomplicated hemorrhage if sufficiently severe, or "traumatic shock" if the loss of whole blood does not seem sufficient to account for circulatory failure. Actually, many cases appear to be varying combinations of sublethal hemorrhage and of traumatic shock.

There is a critical level of blood pressure below which the circulation is inadequate and tissues suffer damage. Damage to the medullary centers from prolonged low pressure probably accounts for loss of vaso motor tone late in shock.

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Paresis or parietic dementia may likewise present convulsive seizures and unconsciousness and the patient may remain in coma for many days. The pupils are small irregular and fixed in paresis. Stolid smooth facies and irregular unequal Argyll Robertson pupils especially with primary optic atrophy tremulousness of the tongue and deep reflex changes may be considered characteristic of general paralysis of the insane. Blood serology and spinal fluid serology are usually positive. The cerebro spinal fluid shows an increase in lymphocytes plasma cells globulin and a typical high parietic gold curve.

### Poisonings

*Poisoning*, if alcoholism is included in this category, will account for about as many cases of coma amenable to cure as any other group of conditions. Poisoning should, therefore, be suspected and considered early in the differential diagnosis of every emergency case. It must be remembered that poisons may be accidental, suicidal, and homicidal. Poisonings may produce laboratory and neurological findings that may be misleading as sugar, albumin, and blood in the urine, and pathological reflexes.

**Opium derivatives** should be suspected as a cause of coma if the breathing is very slow and the pupils are constricted to pin point size.

**Barbiturates** may produce a deep and persisting coma with profound narcosis, complete areflexia, slightly slowed respiration and dilatation of the pupils. Reflexes are depressed. There is positive Babinski, radiokokinesia, asynergia, nystagmus, tremor of the tongue lips and fingers, and convulsions may develop. Encephalitis lethargica, tabo paresis, or multiple sclerosis may be simulated.

**Bromide intoxication** may produce restlessness, confusion, disorientation and hallucination and may be diagnosed by the finding of high levels of bromide in the blood.

**Acute ethyl alcoholic intoxication** is one of the commonest causes of a comatose state and the patient presents rather characteristic suffusion of his facies, stertorous breathing, hiccupping and vomiting with a strong odor of ethanol on a heavy fetid breath.

The odor of alcohol however should not immediately lead to diagnostic conclusions. The examination should be made for other causes or conclusions.

An exact measurement of the alcoholic content in a patient's stomach or his blood may be measured with Dr. W. W. Jettors's instruments or by chemical means (see pp. 260 and 261). Intoxication is questionable with blood levels of 0.05 to 0.15 %, but definite with levels above 0.15 % which are reached by the imbibing of 6 to 7 ounces of alcohol in half an hour. Coma may be present with levels of 0.25 % alcohol or more.

**Methyl alcohol or wood alcohol** produces profound inebriation, delirium, and prolonged coma with formic acid acidosis, cyanosis, labored and elevated respiration, and dilated sluggish pupils. The urine is acid and contains albumin, casts, acetone bodies and formic acid. Methyl alcohol is usually demonstrable in the blood.

**Phenol and cresol** may be recognized by the odor and the blanched mucous membranes and chemical burns about the mouth with faintness, convulsions, and respiratory paralysis.

Carbon tetrachloride particularly when the contaminant carbon bisulfide was present has caused vomiting and abdominal pain. Sulfur dioxide may produce the same symptoms with eye irritation. Conjunctivitis also results from ordinary ammonia refrigerator gas.

Ammonia, bismuth arsenic, and sometimes potassium iodide has produced stomatitis.

Poisoning with bichloride of mercury arsenic lead antimony, digitalis rarely by quinine, aspirin or saline cathartics has produced diarrhea.

Many types of drug intoxication particularly camphor, strychnine, nicotine, and hyperventilation tetany, cause delirium and convulsion.

Arsenic lead carbon monoxide botulism or cyanide poisoning may result in paralysis.

Cyanide used in the cleaning of silverware in hotels (dip polish for silver especially forks) may cause contamination and produce severe gastroenteritis simulating food poisoning.

Poisoning by food is very common and often causes very severe gastro intestinal symptoms such as nausea vomiting and diarrhea.

The clinical picture may give also the suggestion of food contaminated by staphylococci by streptococci or salmonella. These would give rise to acute gastroenteritis nausea vomiting and diarrhea.

**Bacterial Food Poisoning**—Staphylococcal food poisoning comes on within 1 to 6 hours produced by the enterotoxin of the staphylococci yielding an intoxication type of poisoning causing vomiting abdominal cramp nausea and diarrhea.

Salmonella food poisoning is produced by one of the following agent *S. enteritidis* *S. typhimurium* *S. choleraesuis* or others producing an infection type of food poisoning within 7 to 72 hours. The common symptoms are abdominal pain diarrhea chills fever and vomiting.

Streptococcus food poisoning caused by alpha streptococci comes on within 5 to 18 hours causing nausea colic and diarrhea.

Botulism caused by *Clostridium botulinum* produces an intoxication simulating encephalitis in from 18 hours to 3 to 4 days. It causes such symptoms as palsies of the pharyngeal ocular laryngeal and respiratory muscles dysphagia diplopia aphonia and respiratory difficulty as stertorous breathing.

### Acute Overwhelming Infections Producing Syncope and Coma

Meningitis as the cause of coma is easily recognized by the presence of a stiff neck and positive Kernig's and Brudzinski's signs except in infants. The presence of any of these calls for an immediate lumbar puncture and examination of the spinal fluid for bacteria and cells. Cul-

tures should be started before any type of therapy is instituted. The cerebrospinal fluid pressure should be measured. Protein, sugar and chloride content of the spinal fluid may be of significance in diagnosis.

**Pneumonia** may cause coma so severe that shock and cerebral anemia may occur. The fever is usually high. Characteristic signs are usually present in the lungs. If there is no sputum or pharyngeal exudate from which cultures or smears can be made, it is justifiable to puncture the infiltrated lung and get the material for culture and smears. If pneumococci are present in the sputum, they should be typed if specific serum is available. Chest films should be made in all cases of suspected lung involvement and in every patient with fever.

The malignant cerebral type of *estivo autumnal malaria* should be suspected if there have been chills and fever and the patient in delirium has indicated such, particularly if he comes from a malarial district. Blood smears should be made, stained with the usual stain for malaria and the double small ring forms should be looked for. The crescent forms rarely appear in the peripheral blood smear.

**Pulmonary embolism** is often so severely shocking as to cause syncope and the electrocardiographic findings may be fairly characteristic with a picture of acute right ventricular dilatation, but may be indistinguishable from that of acute coronary occlusion with myocardial infarction.

The overactivity of the pulmonary conus and the presence of a pulmonary friction rub, dyspnea, and cyanosis are suggestive signs especially in a recent parturient mother, an orthopedic patient, or a patient with heart failure or an extensive infarction.

### Metabolic Coma

Metabolic coma due to abnormal metabolites is a most common medical emergency.

**Diabetic coma** with acidosis is a common general cause of the comatose state. Usually the patient shows a deep Kussmaul type of breathing with a fruity odor to the breath which is usually considered to be the result of the exhalation of acetone bodies. The eyeballs are soft and the pupils are moderately dilated. The blood pressure is usually low but in elderly diabetic patients it may be elevated. No paralyses or neck rigidity are present.

Usually the urine shows a reaction for ketone bodies, beta hydroxybutyric acid, diacetic acid or acetone. Although sugar is usually present in the urine, it may be absent. Blood should be taken for the determination of the ketonemia, CO combining power, blood sugar and non

protein nitrogen level if facilities are available for these chemical analyses. Extreme hypoglycemia may be the cause of coma and should be ruled out in such cases before insulin is given. The blood sugar determination is highly desirable for differentiation particularly when acetone bodies are not found in abundance in the urine.

*Hypoglycemia* as a cause of coma is a rather rare condition except in the case of hyperinsulinism in diabetic patients. This condition is readily recognized on both clinical and laboratory findings. The pupils are dilated, the eyeballs firm, and the skin is usually wet and shows evidence of pallor. A positive Babinski is present.

The finding of sugar in the first specimen of urine is often misleading, as the bladder may contain urine voided while the patient was hyperglycemic. Repeated urine specimens should be tested for sugar and acetone bodies. A blood sugar should be taken at once for verification of the condition of insulin shock.

*Uremia* is to be suspected in the comatose patient when there is a urinous odor to the breath, unusual dryness to the skin, and elevation of the blood pressure with or without Cheyne Stokes breathing. There may be a uremic frost present in the more severe case of uremia. The heart is usually enlarged, the eyegrounds show evidence of the arterial disease such as hemorrhages and exudate. Pericarditis may develop. The abdomen is usually scaphoid. No paralysis is present in the usual case.

*Acidosis* is present as a rule because of the disturbance of the acid base balance incident to loss of fixed base. Albumin may be present in the urine but not necessarily in large quantities. Occasionally casts are noted in the urinary sediment. A low specific gravity is characteristic. Blood chemical studies reveal an elevation or the retention of the nitrogenous waste products with an elevation of the nonprotein nitrogen, urea, uric acid, and creatinine.

*Eclampsia* is the diagnosis when the patient is a pregnant female with the above findings.

*Cholemia* may be suspected in the patient with the above symptoms and ascites in whom the liver cannot be felt. Coma would then be the result of hepatic rather than renal insufficiency. The blood and the urine would not show evidences of renal disease.

### Syncope and Coma Due to Cardiovascular Disease

Cardiovascular conditions are to be found at the top in statistical studies of the causes of syncope and coma.

Of the local circulatory disturbances which cause syncope, cerebral hemorrhage heads the list. Other local circulatory disturbances are cere



bral thrombosis, hypertensive encephalopathy, cerebral embolism, and cerebral angiospasm. The general circulatory disorders causing syncope are ordinary splanchnic faints, ruptured aortic aneurysm, hemorrhage resulting in shock, postural hypotension, and pulmonary embolism.

*Apoplexy or cerebral hemorrhage* is perhaps the most common single cause of unconsciousness. Convulsions rarely occur except when a cortical artery is involved. The face is usually flushed, the pulse slow, full and tense, and the blood pressure increased. The eyeballs are fixed or deviated to the paralyzed side presenting the so called conjugate deviation. The pupils are contracted and do not react to light. The mouth may be drawn to one side as a result of the paralysis present on the opposite side. Breathing may be Cheyne Stokes in type. Although the reflexes may be flaccid at first, within a few days they become spastic, and abnormal plantar reflexes appear. The coma may only last for a few hours but is rarely of short duration usually lasting for several days. Fever may be present. The spinal fluid may or may not be bloody.

*Subarachnoid hemorrhage* is suspected when there is a gradually increasing stupor, coma, nuchal rigidity, headache, and vomiting. Lumbar puncture usually yields a spinal fluid which is bloody. Removal of a small quantity of the cerebrospinal fluid usually decreases the pressure and relieves the symptoms. An increase in intracranial pressure may be evidenced by papilledema visible on ophthalmoscopic examination of the ocular fundus. Abducent palsy is not an uncommon finding. There are no significant reflex changes and no general paralysis.

*Cerebral embolism* occurs most frequently in patients with acute endocarditis and valvulitis or chronic mitral stenosis, but occasionally may occur also after coronary occlusion. If the embolus is a large one syncope and coma may result. Convulsions occur if the circulation in the cortex is interrupted. Usually the blood pressure is not increased. Unless the embolus strikes the central retinal artery there will be no eyeground changes. The finding of disease in the heart, as well as a febrile condition, makes the diagnosis most likely.

*Cerebral angiospasm* usually occurs in hypertensive patients. Definite changes may be found in the retinal arteries during the attack, silver wire arterioles with irregular and luminal narrowing segmental in type. Transient paralysis may appear and clear overnight. Visual disturbances are common and may persist. Since edema of the brain is usually present there may be an increase in the intracranial pressure. Both systolic and diastolic blood pressures are elevated.

*Hypertensive encephalopathy* presents similar findings to cerebral angiospasm. The syncopal attack may be preceded by headache, visual disturbances, nausea, vomiting and rising blood pressure levels. In

oscopic examination of the eyes reveals evidences of vascular disease constricted tortuous arterioles arteriovenous nicking hemorrhages or exudate and edema of the nerve heads with venous congestion. One may have difficulty in differentiating this fundus picture from that of intracranial neoplasm. Peripheral and renal evidences of hypertensive arteriolar diseases direct the careful examiner to the correct diagnosis.

General cardiovascular conditions with low blood pressure account for relatively few attacks of syncope and coma; however, the recognition of these disorders is necessary. Hypotension and cerebral anemia sometimes result from such acute vascular accidents as pulmonary embolism dissecting aortic aneurysm coronary thrombosis rupture of an aneurysm or of the heart or an aortic cusp.

### Cardiac Syncope

Cardiac mechanism disorders may result in hypotension cerebral anemia and syncope. Ventricular asystole produces Adams Stokes syndrome of convulsive syncope. The diagnosis is suggested by the fact that no heart sounds are audible no pulse is present sharp vertigo and syncope develop rapidly and convulsions often follow.

Paroxysmal ventricular fibrillation may be indistinguishable with out a concomitant electrocardiogram in rare cases from Adams Stokes attacks.

Paroxysmal and atrial flutter or tachycardia especially the one to one type and high grade atrial fibrillation occasionally cause syncope.

Excessive carotid sinus irritability with cardiac standstill or with vagotonia may account for loss of consciousness. Nitrite overdosage postural or orthostatic hypotension and neurocirculatory asthenia are occasional causes of cerebral anemia. Mitral stenosis with a ball valve thrombus or cardiac tamponade of traumatic or hemorrhagic origin or a result of acute pericarditis will account for occasional instances of syncope. Ordinary splanchnic faints although very common are rarely seen by the physician.

Coronary occlusion with myocardial infarction may occasionally produce such great cardiovascular shock and forward failure as to result in syncope. The pain is excruciating and persistent usually substernal or epigastric in location. The blood pressure usually falls abruptly and to critically low levels. There is usually extreme dyspnea. A friction rub may appear and the electrocardiogram is usually striking with an RST elevation and beginning T wave negativity. The fluoroscopic examination may show defective movements of the infarcted myocardium, leukocytosis and an increased erythrocyte sedimentation rate.

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## Chapter XXVII

### MANAGEMENT AND TREATMENT IN EMERGENCY SITUATIONS

Emergency disorders often develop suddenly to the point where life is in the balance and proper therapeutic steps must be taken to turn the tide. Clear vision, unfailing judgment, prompt action, quick diagnosis, and unwavering decision must be exercised. Hemorrhage must be stopped if it is possible to do so. Shock and infections must be combated. Poisons must be removed or neutralized. Pain must be promptly relieved if it is severe but narcotics should not be given to a patient with a head injury or to a comatose individual. Consciousness should be restored as soon as possible. Blood pressure and heart action must be re-established. Delirium and convulsions must be controlled by sedatives. Depression must be relieved by stimulants. The air passages must be cleared and kept open. Respiration must be restored by artificial means and oxygen added by mask if there is the slightest tinge of cyanosis. Life must be sustained during the crises.

#### Management of Trauma to the Head

Almost all injuries to the head are medical emergencies. After x-ray films of the skull have been taken and interpreted the patient should be observed and events noted. In many cases surgical assistance is needed as in compound fractures of the skull.

Consciousness is usually restored spontaneously. Analgesics are given if the pain is great. Hydration must be maintained by intravenous fluids during the period of unconsciousness especially if it is prolonged. Adequate nursing care is particularly important in head injuries.

#### Treatment of Epilepsy

Because continuous fits may cause death from exhaustion sodium phenobarbital or Amytal 0.2 to 0.4 Gm (3 to 6 gr) should be given intramuscularly. Doses of 0.2 Gm (3 gr) every 3 hours may be repeated as necessary. Paraldehyde or Avertin by rectum will be effective if phenobarbital is ineffectual.

#### Management in Brain Tumor or Edema

In cerebral tumor the diagnosis is usually made before coma results however if the patient comes in unconscious and there is papilledema the emergency injection of hypertonic 50 % glucose or sucrose up to 50 cc may be given slowly.

Massive pulmonary embolism usually not found for a few days has been the cause of syncope in recent post partum women. Besides occurring in the complicated puerperium, it often follows cesarean section, pelvic and appendiceal operations, congestive heart failure, or in patients who have been bedridden or have a transmural right infarction. Teofilo Ortiz Ramirez noted early an interscapular souffle of a pulmonary artery partially obstructed by saddle embolus while the lung fields remained clear.

Sudden acute left ventricular failure is recognized by hypertension, aortic regurgitation, orthopnea, Cheyne Stokes breathing, paroxysmal nocturnal dyspnea, pulmonary edema with rales, anxiety, air hunger, a cough productive of pink frothy sputum, and usually a tracheal rattle.

Acute right ventricular heart failure is characterized by exhaustion, engorged neck veins and liver, abdominal pain, distention, and ascites, pedal edema, hydrothorax, general anasarca and dyspnea.

Dissecting aneurysm of the aorta usually results in an extreme and precipitous drop in blood pressure and a shock state. The pain is deep, boring and excruciating in the back and radiates downward and often into the legs. Fluoroscopy and radiograms will show a broadened mediastinal shadow of the aorta. Dissection may extend back to the root of the aorta and around a coronary artery to compress it and produce characteristic ECG changes, or into the aortic valve resulting in incompetency, and into the pericardium where a friction rub is heard. The dissection may extend around the renal arteries of the iliaes and compress to the point of obstruction.

such as black coffee or caffeine orally or by rectum, caffeine sodium benzoate or nikethamide (Coramine) are useful. Forcing fluids with soft foods to maintain strength is necessary. Intravenous injections of 50 cc of 50 % hypertonic glucose 500 cc of 10 % glucose 1 000 cc of 5 % glucose to which may be added various vitamins in the usual doses particularly thiamin 50 mg niacin 100 mg, and insulin 10 to 20 units are all in order to increase the rate of alcohol oxidation in the body.

Cases of methyl alcohol intoxication should be given 1 teaspoonful of baking soda sodium bicarbonate in a glass of water, repeated every hour. In some cases use gastric lavage with a 2 % solution of sodium bicarbonate in large quantities. The carbon dioxide combining power should be watched. If it increases a solution of  $\frac{1}{10}$  M sodium lactate or intravenous sodium bicarbonate may be given. Supportive measures are often necessary such as stimulants. An emetic of warm water containing salt or soap solution is in order as are also cathartics of magnesium sulfate or sodium sulfate in doses of a teaspoonful in warm water. Use colonic irrigation with saline as high as possible to remove the poison from the gut.

In poisoning from barbituric acids an adsorbent and emetic should be used such as charcoal in warm water if the cooperation from the patient can be had. A saline cathartic should be given through a stomach or duodenal tube. Oxygen and carbon dioxide are to be given if there is cyanosis. Analeptics as nikethamide (Coramine) in 5 cc doses Metrazol or picrotoxin must be given until muscular spasm occurs or good respirations persist. If the blood pressure drops add ephedrine sulfate. With cerebral edema and increased intracranial pressure a lumbar puncture should be carried out to lower the pressure of the cerebrospinal fluid. If the respiration gets down to 4 per minute atropine should be given intravenously 0.005 Gm and artificial respiration instituted. Mechanical stimuli ammonia inhalation repeated lavage of the stomach using charcoal in water or a dilute solution of iodine or potassium permanganate 1:2 000 should be continued.

Roach poisons containing phosphorus call for gastric lavage with dilute potassium permanganate solution or copper sulfate solution white of eggs beaten in milk gelatin no oil except mineral oil and saline cathartics. Methionine crystals, 10 Gm in 3 % solution that has been autoclaved may be given intravenously or by mouth with or without calcium lactate to protect the liver cells.

Insect powders containing fluoride call for gastric lavage with calcium salts such as lime water 1 % solution or large quantities of milk.

### Treatment in Bulbar and Upper Cord Poliomyelitis

Serious emergency situations develop when poliomyelitis involves the cranial nuclei. Involvement of the glossopharyngeal nerve results in disturbances in talking and in swallowing with further complicating obstruction of the airways, and asphyxia. Special efforts must be put forth constantly to keep the airways open by continuous suction, postural drainage, intubation, and with tracheotomy and antibiotics.

If the respiratory and circulatory centers in the medulla are affected, these vital functions may fail and lead to a very rapid development of hypoxia. This calls for immediate and augmented delivery of oxygen to the lung by pressure mask or through a tracheotomy tube.

A respirator should be in readiness and when the upper spinal cord as well as the medulla are involved the patient should be tracheotomized and put in the machine. He must be instructed as to the effects of the respirator. The airways must be kept open constantly with no interruption as forced inspiration against obstruction may precipitate pulmonary edema.

The oxygenation of the blood must be maintained. An oximeter of Millikin type (Waters Conley, Rochester Minnesota) will greatly facilitate this and help to keep the  $O_2$  saturation constant and adequate.

### The Therapy of Poisoning

In cases of poisoning the patient usually vomits or induces vomiting. If the patient has not vomited, vomiting should be encouraged or induced by mechanical means so as to remove as much of the poison as possible. The old fashioned, slightly large and stiff stomach tube is preferable and should be carefully introduced to prevent mucosal injury. If the poison has been a heavy metal, a 10 cc dose of 10 % 2,3 dimercaptopropanol (BAL) should be introduced into the stomach.

The patient also should be given an emetic after taking skimmed milk combined with the white of eggs with or without gelatin powdered charcoal, and after alkali or acid according to the indication has been introduced into the stomach. Warm tea is not only a stimulant, but tannic acid in a strong solution would take care of some alkali. Acids may be neutralized by magnesium or thin aluminum gel.

Lysol, phenol and cresol burns may be removed by washing with dilute alcohol or 40 proof whisky. Order the patient to lavage with this gargle, swallow, and then regurgitate it. As demulcents vegetable oils salad oils or cooking oil starch solution, flour water, or thin mashed potatoes act for the protection of the mucous membranes.

**Acute ethyl alcohol intoxication** Lavage the stomach with warm tap water. Apomorphine may be given to induce vomiting. Stimulants

such as black coffee or caffeine, orally or by rectum caffeine sodium benzoate or mkethamide (Coramine) are useful. Forcing fluids with soft foods to maintain strength is necessary. Intravenous injections of 50 cc of 50 % hypertonic glucose 500 cc of 10 % glucose 1000 cc of 5 % glucose to which may be added various vitamins in the usual doses particularly thiamin 50 mg niacin 100 mg and insulin 10 to 20 units are all in order to increase the rate of alcohol oxidation in the body.

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Insect powders containing fluoride call for gastric lavage with calcium salts such as lime water 1 % solution or large quantities of milk.



Calcium may be given intravenously as calcium gluconate or calcium chloride. Saline cathartics and antishock methods are generally necessary supportive treatment.

**Rat poison** usually contains cyanide. It makes for difficult respiration. Oxygen usually is necessary, as a forced measure with a respirator. Amyl nitrate, inhalation, emetics, gastric lavage with 10 % sodium thiosulfate or 3 % hydrogen peroxide sodium thiosulfate 25 % intravenously, or 50 cc of 1 % sodium nitrite can be given intravenously. Every effort should be made to prevent shock and collapse by general supportive measures.

**Ant poison** contains thallium salts. It should be treated with emetics and saline cathartics. There should be complete gastric lavage. A dessertspoonful of charcoal powder 2 parts of 1 part magnesium oxide and 1 part tannic acid given as an emetic should be brought up by stomach washing and then a second dose administered. Besides the emetic and stomach pumping, the patient should be given Ringer's solution and glucose intravenously. Shock should be treated by the usual medical means. Continued vomiting and diarrhea result in the loss of electrolytes, chloride and fluids and can cause collapse. This condition calls for oral lactate buffer solution to be given in repeated doses, 5 cc diluted 10 times, and as often as every hour.

### **The Treatment of Arsenical, Mercury, and Other Metallic Poisonings With BAL (British Anti Lewisite)**

The dithiol BAL, 2,3 dimercaptopropanol, was found to form a compound with arsenic that was nontoxic and more stable than compounds made with monothiols cysteine or glutathione. The heavy metals, particularly arsenic and mercury, are prevented from attaching themselves to the sulfhydryl groups of the cellular proteins and thus preventing the interference with the essential enzyme systems which require free sulfhydryl groupings for their action.

BAL was developed during the World War II in Peters' Laboratory in Oxford by Stocken and Thompson. It is a nontoxic and nonirritating preparation that can be used for intramuscular injection, and is supplied in sealed ampules of 5 % or 10 % BAL in 20 % benzyl benzoate in peanut oil. Intramuscular injection of 3 mg per kilogram of 10 % solution produced hardly any symptoms, was rapidly excreted, and could be repeated every 4 hours. 5 mg per kilogram produced nausea, vomiting, headaches, burning sensation of the lips and mouth, conjunctivitis, salivation, constriction of the throat, chest and hands, tingling sensation

in the hands burning sensation in the penis sweating of the forehead and hands abdominal pains tremors and shakiness, elevation of the blood pressure

These toxic symptoms could be relieved by injection of 0.6 cc 1:1000 epinephrine solution or prevented by an oral dose of 25 to 50 mg of ephedrine  $2\frac{1}{2}$  hours before injection. Exfoliative dermatitis of arsenical origin responded in 70 % to 80 % of the cases, some of them within five days and most of them by the fifteenth day. BAL has been effective in arsenical hemorrhagic encephalitis optic neuritis blood dyscrasia agranulocytosis and thrombocytopenic purpura.

Mercury bichloride poisoning was found to respond experimentally and clinically if BAL was administered within 15 to 30 minutes. BAL has been effective when administered orally as long as 3 hours after ingestion. BAL was found by Thompson and Whitaker to protect the pyruvate oxidative system of the brain from toxic effects of mercury. Nausea vomiting hematemesis melena diarrhea and abdominal pains were relieved within 24 to 48 hours. Severely poisoned cases were completely restored within three or four days in the majority of instances. Diarrhea anemia ulceration of the mucous membrane and renal insufficiency rarely persisted. Treatment started within 4 hours saved most patients who had taken 1 to 20 Gm of  $\text{HgCl}_2$ . In acute poisoning the initial dose is usually 300 mg which amounts to 5 mg per kilogram followed in 2 to 4 hours by 150 mg. In severe cases this is repeated at 4 hour intervals for 24 to 36 hours. Patients have received as much as 600 to 750 mg during the first 12 hours. The dose is rapidly reduced as the symptoms clear. Continue the maintenance dose of 150 to 300 mg a day for fifteen days.

Antimony poisoning in animals experimentally has been beneficially effected by BAL. In lead poisoning BAL usually augments the toxic action. BAL has no beneficial effects upon the symptoms of lead poisoning in human beings even though the urinary excretion of lead has been increased.

Cadmium forms the compound with BAL in vitro that is readily dissociable and as toxic as cadmium itself. The dissociation usually takes place in the kidneys and produces widespread damage. In polyneuritis cases it has been recorded that improvement has followed BAL therapy. BAL is recorded to have a beneficial effect upon infectious and metabolic neuropathies. Exfoliative dermatitis bone marrow granulocyte depression in patients with arthritis treated by gold salts have been relieved by BAL.

Reference Longcope W. T. and Luetscher J. A. The Use of BAL. *Ann Int Med* 31: 545, Oct. 1949

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### The Treatment of Arsenical, Mercury, and Other Metallic Poisonings With BAL (British Anti Lewisite)

The dithiol BAL, 2,3 dimercaptopropanol, was found to form a compound with arsenic that was nontoxic and more stable than compounds made with monothiols cysteine or glutathione. The heavy metals particularly arsenic and mercury, are prevented from attaching themselves to the sulfhydryl groups of the cellular proteins and thus preventing the interference with the essential enzyme systems which require free sulfhydryl groupings for their action.

BAL was developed during the World War II in Peters' Laboratory in Oxford by Stocken and Thompson. It is a nontoxic and nonirritating preparation that can be used for intramuscular injection and is supplied in sealed ampules of 5 % or 10 % BAL in 20 % benzyl benzoate in peanut oil. Intramuscular injection of 3 mg per kilogram of 10 % solution produced hardly any symptoms, was rapidly excreted, and could be repeated every 4 hours. 5 mg per kilogram produced nausea, vomiting, headaches, burning sensation of the lips and mouth, conjunctivitis, salivation, constriction of the throat, chest and hands, tingling sensation

only daily for a week. There follows prompt relief of pain, dyspnea, orthopnea, cyanosis and often a clearing of the comatose state.

The treatment of cerebral thrombosis and embolism calls for heparin in at least 50 mg doses given intravenously every 4 hours or continuously by intravenous drip; intramuscular Depoheparin. The clotting time is to be repeated every 2 to 3 hours and should not be more than 60 minutes. Dicumarol may be started with a 100 mg dose and continued with 100 mg doses at 24 and 48 hours. Prothrombin time should not be progressed more than 60 % of normal. The prothrombin time should be taken at 12 hour intervals.

Toluidine blue or protamine sulfate antagonize the action of heparin and vitamin K that of Dicumarol in hemorrhagic states. Whole blood transfusions are preferable to stop hemorrhage from thrombocytopenia or neutropenia that results from radiation or occurs spontaneously.

### Treatment of Diabetic Acidosis and Coma

Draw blood for sugar, acetone, CO<sub>2</sub> and N.P.N. determinations. Start saline intravenously without delay.

According to Joslin, severe ketosis exists when the carbon dioxide combining power falls below 20 vol %. Insulin in sufficient dosage at least 50 units subcutaneously immediately to correct the ketosis and hyperglycemia is the essential in the treatment of this condition. There are other important therapeutic measures which will be discussed later.

The urine and blood sugar level as well as the acetone or ketone level should be determined at least every hour during the emergency state or until the blood sugar drops below 200 mg %, and then in 2, 4, 8 and 12 hours. The CO<sub>2</sub> combining power is determined every 3 hours.

Joslin advocates regular or crystalline insulin in the emergency; however, protamine zinc insulin is approved by other specialists in the field of diabetes. The average dose of insulin varies from 50 to 100 units of regular or crystalline insulin given subcutaneously or intravenously.

Acidotic patients require 50 units subcutaneously at the outset. If the patient is unconscious and in shock, a similar dose must be given intravenously. One half hour later give a second dose of insulin of the same size. Usually two other doses of insulin of 50 units each will be needed at half hour intervals depending upon the original blood sugar, carbon-dioxide combining power and ketone bodies in the urine and the response to the therapy.

At the end of about 2 hours another specimen is taken for blood sugar and carbon dioxide combining power. Insulin is then given according to the results on these tests. It may be necessary to give as

much as a 100 unit dose of insulin at 30 minute or 1 hour intervals. When the patient is able to void hourly, the administration of insulin may be gauged according to the results of the urine sugar. Inject 24 units for a 4 plus, 20 units for 3 plus, 16 units for 2 plus, and 12 units for 1 plus.

As the patient's condition improves, the interval for collection of specimens and the giving of insulin may be increased to from 2 to 4 hours. 1,000 or more units of insulin may be needed in the first 6 hours. After the ketosis is well controlled, the patient may be gradually shifted back to his regular diet and insulin regime. As soon as fluid can be taken by mouth, this route should be used. The average case will require 4 to 5 liters of fluids in the first 24 hours.

Since most patients in diabetic coma are dehydrated and their store of electrolytes is low, the use of isotonic fluids is advocated. There is a difference of opinion as to what fluids are best for intravenous use. Normal saline is probably preferable as it corrects dehydration, hemoconcentration and loss of chloride without the disadvantage that glucose has of masking the effect of the first dose of insulin. Lactated Ringer's solution (Hartmann) or 1/6 M sodium lactate solution may be used. Usually 1 liter to 1.5 liters should be started intravenously immediately and repeated according to the state of hydration in 6 hours.

If acidosis is very marked in proportion to the blood sugar level, Joslin will permit the use of glucose plus the saline solution since the ketosis is what threatens the patient's life, not the hyperglycemia.

Some authorities recommend the use of *alkalies* in patients with severe acidosis and a carbon dioxide combining power below 15 vol. %. Duncan gives 300 cc. of a 3 % solution of sodium bicarbonate or 1/6 M sodium lactate solution. If giving sodium lactate the formula for restoring the carbon dioxide combining power to normal levels is  $(40 \text{ plasma content of CO}_2) \times (0.3 \times \text{body weight in kilograms}) \times 5$ .

The blood serum potassium level should be determined or an electrocardiogram taken, and if there is a subnormal level or hypokalemia or ECG signs thereof, steps should be taken to relieve the deficiency as outlined under potassium salts intravenously.

Four to six hours after insulin has been started, diet usually may be started in the form of fluids: orange juice, broth, tea, etc. After fluids are well tolerated, soft diabetic foods may be begun.

General measures such as keeping the patient warm by the use of blankets or artificial warmth, should be used. It is advisable to wash out the rectum with warm water early in the treatment of diabetic coma. If abdominal distention is present, an enema may be given.

### Management of Hypoglycemia

Because this condition may be confused with diabetic coma and insulin administered it is advisable to obtain the level of blood sugar before any specific treatment is started.

When symptoms of hypoglycemia begin the patient should take some rapidly absorbable form of carbohydrate as candy, sweet orange juice, lump sugar or such. If the patient is in coma treatment is urgent.

The comatose patient cannot swallow and so glucose must be given intravenously in doses of 20 to 50 cc of 50 % solution. If sterile glucose is not available, 10 minims of 1:1000 Adrenalin given subcutaneously may be sufficient to revive the patient so that he can be given glucose or sugar by mouth. The necessary adjustments of insulin and diet should be made when the patient regains consciousness and the hypoglycemia has been corrected.

### Treatment of Uremia

The treatment of uremia depends upon the cause. If due to obstruction of the prostate at least palliative surgery is necessary in most cases. In acute nephritis the patient should be kept warm in bed with hot packs over the kidney and well hydrated, but not overloaded with salt and water and carefully protected from exposure. The cause of renal irritation should be treated vigorously as possible and removed if possible. In the normal course of events oliguria or even anuria of eight days duration has been known to give way to a spontaneous return of kidney function.

When the symptoms of uremia are pending therapeutic measures should be started. If the patient is able to eat the diet should be low in protein and high in carbohydrate with plenty of fluids; however if the appetite is poor the patient should be allowed to eat anything that he desires.

When dehydration is severe from vomiting, diarrhea or polyuria sufficient fluids should be given intravenously or by rectum. If edema is present it is wise to restrict completely the sodium chloride intake. Accompanying cardiac decompensation should be treated appropriately.

In cases of uremia due to advanced bilateral disease of the kidneys therapy is purely symptomatic. In the past sweating and elimination through or irrigation of the upper gastrointestinal tract have been used to remove nitrogen waste from the blood. Purgation methods weaken the individual considerably but colonic irrigation with gallons of magnesium sulfate solution may be helpful temporarily. Gastrointestinal tract irrigation through a Wangenstein or a Miller Abbott tube may help some. Clearing the blood of NPN by dialysis and peritoneal lavage has been used experimentally but has not yet proved practical or safe.

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In hopeless cases, sedatives should be used, phenobarbital to prevent vomiting and control excitement. Paraldehyde, chloral hydrate, or morphine may be used to control sleeplessness and restlessness. Barbiturates, chloral, and intravenous magnesium sulfate are indicated if convulsions develop. Lumbar puncture is often useful in relieving severe headaches.

Acidosis may be relieved by sodium bicarbonate by mouth intravenously, or by rectum in appropriate dosage. Calcium gluconate intravenously will prevent muscular twitching.

### Treatment of Cholemia

Since the liver has so many functions various measures must be employed to try to protect each of these. When the liver contains adequate stores of glycogen, it is protected from the action of toxins. This glycogen is provided by glucose given intravenously 0.25 Gm per kilo gram. Because insulin aids the deposition of glycogen, it may be given in doses of 10 units for every 50 Gm of glucose. The fluid intake should be increased. Drainage of the biliary tract, duodenal and colonic irrigations with hypertonic magnesium sulfate solution may remove some toxic waste products. Injections intravenously of glucose solution containing Hartmann's sodium lactated Ringer's solution will usually bring the patient out of the comatose state temporarily.

Vitamin B complex and vitamin K are useful. Methionine and choline given in doses of 6 to 10 Gm intravenously or orally and in preventing the advance of severe liver damage and may cause the mobilization of excessive fatty deposition from the liver and improvement.

### Emergency Treatment in Acute Cardiovascular Episodes and Heart Attacks

*A benign attack of syncope, splanchnic congestion cerebral anemia* or ordinary fainting is suggested by pallor, slight cyanosis, rapid feeble pulse, and low blood pressure. The patient's head should be dropped lower than the rest of his body if he has not already fallen to the floor. Cold water dashed to the face helps to revive the patient.

*Carotid sinus or vasovagal fainting or sino atrial standstill* causing a momentary ventricular standstill is usually precipitated by pressure on the neck. By the time the patient is examined, he has often recovered. The pulse is slow. The blood pressure is low. He may or may not have fallen out. In such patients pressure on the carotid sinus usually stops the heart momentarily and reproduces a syncopal attack. Removal of pressure on the neck is all that is usually necessary. Intravenous injection or oral administration of atropine 1.0 mg ( $\frac{1}{6}$  gr), or tincture of

belladonna by mouth in the usual dosage of 5 to 10 drops decreases sensitivity of the vasovagal reflex and usually prevents attacks

Orthostatic postural hypotension is a vasovagathetic disorder coming on after the patient has been standing in an upright position for 3 to 4 to 5 minutes. There is gradual development of slight giddiness, vertigo, and decreased pulse rate as the systemic blood pressure drops to critically low levels. Ephedrine or benzedrine usually restores these patients. Paredrine and desoxycorticosterone are temporarily effective in maintaining blood pressure levels on standing.

### Heart Attacks Associated With Palpitation

In rare cases rapid heart action results in syncope. Paroxysmal atrial or supraventricular tachycardia, a benign disorder of the heart mechanism, may sometimes be serious. It may occur in perfectly normal individuals postoperatively or during infection and be very disturbing. The heart rate may jump suddenly within one beat from 85 to 180, even up to 240 beats per minute and the patient feels quite tense. The tachycardia may be momentary, last a few minutes or go on for hours. It is regular in rhythm with rate exactly the same from minute to minute. There may be sudden offset within one beat to critically low bradycardia.

The treatment is first to try the carotid sinus pressure or the oculocardiac reflex. The patient should lie down bending over forcing inspiration or expiration against the closed glottis. Half of the attacks are stopped with carotid sinus pressure. A quick acting barbiturate, as Seconal or Delvinol may be used to advantage but rarely is it necessary to use morphine. When persistent Mecholyl, magnesium sulfate, digitalis, quinidine, Neo synephrine or Prostigmine by injection will stop the attacks.

Attack may usually be stopped in 35 to 70 seconds by rapid intravenous injection of 0.5 mg of Neo synephrine. If the tachycardia does not subside a larger dose 1 mg at the most may be given when the blood pressure returns to normal.

Attacks may later be prevented by the increase in the diet of potassium containing foods or the administration in small dose of potassium iodide and/or a sedative as phenobarbital. Sometimes quinidine or digitalis must be regularly administered in severe cases. In the patient with myocardial damage who has been digitalized, paroxysmal atrial tachycardia is a serious disorder.

Paroxysmal sinus tachycardia is much rarer but has many of the same characteristics as atrial tachycardia and may be treated in the same way.

**Transient or paroxysmal atrial fibrillation** may present sudden onset and sudden offset and may recur. The rhythm is absolutely irregular, the apical or heart rate is usually high, 140 to 160 per minute. There is a very marked pulsus deficit between apical and radial rates. The usually associated conditions as hyperthyroidism, rheumatic heart disease, arteriosclerosis, coronary artery disease or digitalis intoxication are not necessarily present. Quinidization 0.2 Gm (3 gr) by mouth every hour for 10 doses is usually effective in re-establishing a normal mechanism. Sustained digitalization is also indicated if the high rate and deficit persist or there is evidence of congestive failure present.

**Paroxysmal atrial flutter** also has sudden onset and sudden offset. The patient gives the story of recurrent attacks of rapid regular palpitation, rate between 130 and 150, without any other symptoms. In rare instances a 1:1 mechanism may intervene with a rise in rate to 300 per minute and syncope results, and in which case vagovagal stimulation is indicated. Stimulation of the carotid sinus produces a slowing, increasing the grade of block considerably and slowing the ventricular rate for a short time only, and then the rate rises in spite of continued block. Rapid quinidization or digitalization are usually prescribed. It is sometimes necessary to digitalize to produce fibrillation and after the digitalis effects wear off use quinidine to produce a return to normal rhythm.

**Cardiac tamponade** should be suspected whenever there has been a chest wound and in a chronic cardiac who has had a paracentesis injury or a myocardial infarction or pericarditis. In a patient who has what appears to be acute congestive failure with extreme engorgement of the neck veins, thready pulse, fall in blood pressure, rise in heart rate, indistinct heart sounds and dullness considerably beyond the apex, impulse, pericardial effusion or transudation must be considered. The suggestive signs may be confirmed by fluoroscopic radiology with examination in the erect and supine positions. The therapy consists in pericardial paracentesis and decompression. If whole blood is obtained, surgical exploration is indicated.

**Ball valve thrombus** produces a similar picture with a very thready pulse that may be entirely shut off, a sudden drop in blood pressure, cyanosis and collapse in an individual with a high grade of mitral stenosis, the signs of which may be obscured by the thrombus. Gangrene of the tips of the fingers and of the toes may develop. In the emergency treatment the patient is held in an upside down position and vigorously shaken. This may temporarily dislodge the incarcerated ball thrombus from the mitral valve, however, atriotomy and thromboectomy are heroic but justifiable treatment.

Paroxysmal ventricular tachycardia may occur in occasional normal individuals. It is usually a serious disorder that occurs in patients who have had coronary occlusion with infarction of considerable myocardium of a rather recent date. Characteristic 'P' waves of dissociated slower atrial rate are electrocardiographic signs to indicate the presence of ventricular tachycardia. The rate is not so high 160 to 180 per minute. The rhythm is not absolutely regular but varies 5 to 6 to 8 beats between minute counts. The electrocardiogram of course is diagnostic particularly if there is an identifiable slower atrial rate as evidenced by P waves superimposed in different positions on the R and T waves.

Quinidine sulfate is practically specific medication, 0.2 Gm (3 gr) hourly for 8, 10 or 12 doses. In rare instances the patient's life is jeopardized by persistent refractory ventricular tachycardia and in those intravenous quinidine lactate carefully given under electrocardiographic control is justifiable. Procaine amide in 10% sol is effective.

Adams Stokes syndrome of heart block or ventricular fibrillation origin is usually a recurrent affair of varying grades. In minor as well as major attacks the patients suffer from slight giddiness only or then may fall out completely. They have pallor, grayness, giddiness, vertigo after 5 seconds, syncope after 10 seconds, convulsions after 16 seconds, cyanosis after 20 seconds and death usually after 90 to 120 seconds of asystole.

On examination there usually is a slow rate or there may be a partial block or a complete block with a rate below 40 and as low as 20 per minute. There may be periods of ventricular fibrillation which like the grade of block can be diagnosed with certainty only by electrocardiographic study at the time. There are occasionally also electrocardiograms which will show a sudden change from perfectly normal conduction to complete ventricular asystole within one beat.

Cardiac emergency treatment is directed toward interrupting the ventricular asystole by striking sharp short blows to the precordium until preparation of a syringe with 2 or 3 or 5 minims of 1:1000 epinephrine solution is ready for jugular or intracardiac injection. After jugular injection the patient is to be suddenly pulled into the upright position hoping that the solution will gravitate to the heart. A long needle and syringe is prepared for intracardiac therapy and after the 90 second deadline has passed injection of 3 minims of epinephrine is made as the needle is withdrawn through the heart muscle. This is justifiable heroic therapy.

If runs of ventricular fibrillation are causing the periods of cerebral anemia, procaine amide and quinidine are the only drugs which promise anything in the way of correction. Barium chloride is often given to

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overwhelming attack with extreme dyspnea and syncope. The physical examination may show the conus arteriosus to be overactive. Friction rub may be heard in the pulmonary area. The cor pulmonale picture of the right ventricular strain pattern appears in the electrocardiogram. Later there is an ECG picture that simulates that of coronary occlusion, particularly if the blood pressure is dropped to very low levels.

*Emergency treatment* consists in the injection of 50 mg of heparin, and morphine, papaverine and aminophylline in full dosage as outlined above. Oxygen 100 % 6 liters per minute into an aviator's mask should be given. The Trendelenburg operation for pulmonary embolism is considered justifiable if the patient can be prepared and placed in a special surgical set up.

Massive acute pulmonary embolism is treated heroically if the operating room is set up for the Trendelenburg operation, and operation is certainly justified as the patient gasps the last breath. At the time the diagnosis is made papaverine 0.15 to 0.28 Gm ( $1\frac{1}{2}$  to 3 gr) is given intravenously along with heparin 50 to 100 mg and possibly Priscoline (Ciba). Oxygen should be used to combat anoxia. The source of the embolus should be located and if another develops venous ligation as of the femorals or the vena cava may be necessary.

Peripheral circulatory respiratory collapse arises in an extreme shock state and is brought on after severe trauma to the head. It is indicated by the collapse of the peripheral veins, an almost impalpable thready pulse, distinctly low blood pressure, cyanosis, and extreme weakness and collapse.

Cortin, adrenal cortical extract, desoxycorticosterone acetate, blood plasma, but still better whole blood and the application of external artificial heat are alleviating at times. Even epinephrine may be justifiably used along with caffeine sodium benzoate. A respiratory stimulant as Nikethamide, Metrazol or Coramine, especially in heart attacks that present dyspnea or edema is of value.

Acute congestive heart failure, particularly acute left ventricular failure, presents acute severe dyspnea. There develops orthopnea. It usually occurs in patients with hypertension, aortic regurgitation and after myocardial infarction. Cyanosis, rales and frothy pink expectoration of pulmonary edema may continue producing distress and an anxiety state. Chronic cases show Cheyne Stokes breathing and paroxysmal nocturnal dyspnea.

On physical examination fine subcrepitant rales of pulmonary edema are heard at the bases of the lungs. There may be asthmatic sibilant and sonorous sounds of cardiac asthma which may be difficult to differentiate and which give a clue to the situation.

make the ventricle responsive but, it sometimes makes the ventricle so irritable that the dose must be reduced or it will induce ventricular fibrillation. If convulsive syncopal attacks increase in frequency under barium chloride therapy, the drug should be stopped.

### **Pain Dominated Heart Attacks**

Angina pectoris is paroxysmal acute coronary failure or insufficiency with pain beneath the sternum or across the chest and characteristically radiating down the arm. It usually follows exertion and acute emotional stress, and generally lasts 15 minutes and stops instantly. It may be aborted by the use of nitroglycerin 0.3 mg (1/200 gr) or 0.6 mg (1/100 gr) under the tongue. In hypertensive patients, nitroglycerin is given routinely and often under the tongue to stop the pain, but whisky is desirable if the blood pressure is low. Oxygen by aviator's mask, 6 liters per minute, relieves pain, but it may be used prophylactically. It is best to have the patient avoid precipitating exertion, prevent emotional stress and strain by sedation with phenobarbital, and take coronary vasodilators and vascular alteratives such as niacin 50 to 100 mg and niacinamide 100 to 200 mg tid to prevent or decrease attacks, and aminophylline 0.5 Gm (7½ gr) intravenously or by rectum.

Pain is more persistent in coronary occlusion and myocardial infarction, in which it is usually excruciating, lasts longer than 15 minutes but may be at times surprisingly slight. Urgency in treatment calls for intravenous narcotics: morphine 15 mg (¼ gr) papaverine 65 mg (1 gr), or 20 mg (½ gr) of methadon Pantopon 20 mg (½ gr), or dilaudid 2 mg (⅓₀ gr) or Demerol 65 mg (1 gr). Aminophylline 0.5 Gm (7½ gr) in 10 or 20 cc of solution intravenously is often a coronary pain sedative, when other drugs fail. Oxygen therapy 100 %, 6 liters per minute by aviator's mask is indicated whether or not there is cyanosis. Depo Heparin and Dicumarol 200 mg may be started immediately.

In a dissecting aneurysm of the aorta, the pain and shock may resemble cardiac pain closely, but usually is more widespread propagated down both arms and/or legs. The shock is greater. The electrocardiographic signs of a myocardial infarction are absent until dissection involves the coronary arteries. The treatment is the same, but the shock state requires more supportive measures and only a rare case recovers.

Acute cor pulmonale from acute pulmonary embolism occurs in patients who have just had babies or a surgical operation, phlebitis or chronic congestive heart failure. There is often a slight ominous warning in the recurrences as attacks of dyspnea and great dread of sudden dissolution precede the attacks. There is usually eventually an

ment of the patient will be discussed under management of heart disease by diet, salt and water control digitalis, coronary vasodilators, diuretics, and possible myocardial builders

### Peripheral Vascular Collapse or Medical Shock

Serious symptoms and signs progress rapidly, and unless the pathological processes are not promptly interrupted dissolution may result. The circulating blood volume is reduced and must be combatted by posture warmth, relief of pain replacement and supportive therapy

The reclining posture should be with head low and foot of the bed elevated not more than 15 degrees to shift blood mass to brain and heart. Warmth should be maintained moderately with a light, an electric blanket or electric light cradle. Pain should be relieved by small doses of morphine 8 mg to 11 mg ( $\frac{1}{8}$  to  $\frac{1}{6}$  gr) intravenously with nikethamide as a respiratory stimulant. The excited patient needs reassurance, and a sedative such as Pentothal Sodium intravenously may be necessary. Oxygen under 4 cm of water pressure is the most important single form of medical therapy. Transfusion of blood plasma or better still whole blood at the rate of 200 cc per hour is the most effective blood volume restorative. Normal saline and isotonic glucose are useful but Dextran may be helpful in the absence of blood. In primary shock vasopressor drugs in small doses intravenously may be of some help but in secondary shock these drugs are useless.

### Emergency Management of Acute Coronary Insufficiency or Occlusion

The acute excruciating cardiac pain should be promptly relieved by injecting intravenously slowly papaverine HCl 65 to 130 mg (1 to 2 gr). If this does not relieve give intravenously morphine  $\text{SO}_4$  11 mg or 8 mg ( $\frac{1}{8}$  to  $\frac{1}{6}$  gr). If the patient is sensitive to morphine use Demerol (100 mg) Pantopon (20 mg) or Dilaudid (2 mg) intravenously intramuscularly or subcutaneously.

Oxygen by mask (6 liters per minute as near 100 % as possible) is in order if pain persists if respiration is embarrassed or if cyanosis is present.

Aminophylline 0.5 Gm ( $7\frac{1}{2}$  gr) alone may be given intravenously for status anginosus and 0.2 to 0.5 Gm ( $3\frac{3}{4}$  to  $7\frac{1}{2}$  gr) in 20 cc of 50 % glucose as indicated in the presence of pulmonary edema. Thiomerin 0.1 Gm (1 cc) with ascorbic acid 200 mg may be added to the above and given slowly in the same syringe.

Vasodilators as papaverine aminophylline or niacin should be continued by mouth by rectum intramuscularly or intravenously as necessary.



The emergency treatment consists in the immediate alleviation of anxiety and respiratory distress. Demerol 100 mg intravenously has superseded and is safer than morphine 15 mg and atropine 0.65 mg in cardiac asthma. If exudation into the pulmonary alveoli and the bronchial tree is great, and if the patient is in danger of drowning, a nasal catheter should be inserted and the exudate suctioned off. Oxygen, 100 %, 6 liters per minute in an aviator's mask under pressure of 4 cm of water, will usually relieve cyanosis.

Reduction of the blood volume by venesection or by trapping of the blood in all the extremities by four tourniquets, releasing each one alternately every 15 minutes for 2 to 3 minutes to allow recirculation of the blood mass may be life saving.

Intravenous injection of lanatoside C 0.8 mg, of digitoxin 1 mg, or of ouabain 0.25 mg, has been used successfully as a myocardial stimulant to tone up the chronic and refractory cases with pulmonary edema and cardiac asthma. A heroic mixture of aminophylline 0.5 Gm ( $7\frac{1}{2}$  gr) in 50 cc of 50 % glucose solution and 1 to 2 cc of Thiomerin, with 0.5 Gm of ascorbic acid added, is injected intravenously. If the pulmonary edema recurs, it, of course, should be removed again by diuresis. Precipitating factors should be avoided, and predisposing factors should be corrected and altered in as far as possible. Supportive treatment is usually necessary.

Acute right ventricular congestive failure is less threatening to life except when the patient becomes anasarca. The latter complication of increased salt and water retention is incidental perhaps to the disturbed blood flow through the kidney. The blood volume usually has been greatly increased and the venous pressure elevated. The liver is engorged and greatly distended. Pain over the liver, nausea, vomiting, ascites, water logging of the extremities, edema, hydrothorax, and dyspnea may develop.

Venesection with the removal of 500 cc of blood, under sterile conditions so that it can be used again, is less often practiced than it should be. Paracentesis into the pleural or peritoneal cavities and for the removal of transudated fluid is desirable.

The intravenous use of mercurial diuretics as 2 cc of Mercuzanthin or Salyrgan. Theophylline has been replaced by the intramuscular use of Mercuhydrin, 2 cc, but preferably by the subcutaneous use of Thiomerin, 2 cc. All of these usually produce a profuse diuresis and sharp reduction of edema and body weight.

Diuresis may be augmented by having the patient take adequate doses of ammonium chloride, ammonium nitrate or potassium salts for a few days before injection of the mercurial diuretic. Further manage-

ment of the patient will be discussed under management of heart disease by diet salt and water control digitalis coronary vasodilators diuretics and possible myocardial builders

### Peripheral Vascular Collapse or Medical Shock

Serious symptoms and signs progress rapidly and unless the pathological processes are not promptly interrupted, dissolution may result. The circulating blood volume is reduced and must be combatted by posture warmth relief of pain replacement, and supportive therapy.

The reclining posture should be with head low and foot of the bed elevated not more than 15 degrees to shift blood mass to brain and heart. Warmth should be maintained moderately with a light an electric blanket or electric light cradle. Pain should be relieved by small doses of morphine 8 mg to 11 mg ( $\frac{1}{8}$  to  $\frac{1}{6}$  gr) intravenously with nikethamide as a respiratory stimulant. The excited patient needs reassurance and a sedative such as Pentothal Sodium intravenously may be necessary. Oxygen under 4 cm of water pressure is the most important single form of medical therapy. Transfusion of blood plasma or better still whole blood, at the rate of 200 cc per hour is the most effective blood volume restorative. Normal saline and isotonic glucose are useful but Dextran may be helpful in the absence of blood. In primary shock vasopressor drugs in small doses intravenously may be of some help but in secondary shock these drugs are useless.

### Emergency Management of Acute Coronary Insufficiency or Occlusion

The acute excruciating cardiac pain should be promptly relieved by injecting intravenously slowly papaverine HCl 60 to 130 mg (1 to 2 gr). If this does not relieve, give intravenously morphine SO<sub>4</sub> 11 mg or 8 mg ( $\frac{1}{6}$  to  $\frac{1}{8}$  gr). If the patient is sensitive to morphine use Demerol (100 mg) Pantopon (20 mg) or Dilaudid (2 mg) intravenously intramuscularly or subcutaneously.

Oxygen by mask (6 liters per minute as near 100 % as possible) is in order if pain persists if respiration is embarrassed, or if cyanosis is present.

Aminophylline 0.5 Gm ( $7\frac{1}{2}$  gr) alone may be given intravenously for status anginosus and 0.25 to 0.5 Gm ( $3\frac{3}{4}$  to  $7\frac{1}{2}$  gr) in 20 cc of 50 % glucose as indicated in the presence of pulmonary edema. Thiomerin 0.1 Gm (1 cc) with ascorbic acid 200 mg may be added to the above and given slowly in the same syringe.

Vasodilators as papaverine aminophylline or niacin should be continued by mouth by rectum intramuscularly or intravenously as necessary.

Atropine  $\text{SO}_4$  0.65 mg (1/100 gr) may be added, particularly if there is distension.

If premature contractions or paroxysms of ventricular tachycardia develop, try, in order, papaverine or morphine intravenously, then quinidine  $\text{SO}_4$  by mouth, then intramuscularly, and, if necessary, by vein.

The patient should be kept at rest with regular shifting of position of his trunk by an attendant. Passive movement and massage centrally of the limbs should be carried out at regular intervals.

As soon as the diagnosis is established the use of anticoagulant therapy is to be seriously considered especially if accurate coagulation time and prothrombin times can be done and if the patient has suffered extreme drops in blood pressure or has had persistent symptoms and signs of a progressing thrombosis, or has had an embolus to any part.

Depo Heparin Sodium in gelatin (Upjohn), 280 mg intramuscularly without epinephrine or ephedrine every 24 hours, is most desirable. Depo Heparin Sodium 100 mg in gelatin solution every 8 hours intramuscularly or 120 to 140 mg every 12 hours intramuscularly has proved efficient. Heparin 200 mg in the new Pitkin menstruum may be used daily. Regular heparin 50 mg in 4 cc solution may be given intravenously and repeated every 3 to 4 hours to hold the clotting time to 45 minutes, or heparin continuously intravenously (100 to 200 mg in 1,000 cc in normal saline 0.9 % solution) 20 to 30 drops per minute may be given for 24 to 36 hours until Dicumarol becomes effective. The blood coagulation time and prothrombin activity must be done every 12 hours and kept at 45 to 60 minutes for blood coagulation and above 20 % and below 25 % for prothrombin activity. If hemorrhage occurs transfusion will usually stop it. Protamine A and toluidin blue will counteract heparin, and vitamin K intravenously usually controls the Dicumarol effects.

Dicumarol may be given orally in one dose of 200 mg immediately and then 100 mg for 1, 2 or 3 days until the prothrombin time drops to 35 % of normal. Then heparin, if given, is stopped and Dicumarol is withheld until the prothrombin time rises to 50 % of normal.

Dicumarol 100 mg, 75 mg, or 50 mg is then given each morning after the prothrombin activity is determined to maintain the level as near to 30 % as possible.

The urine should be watched for the appearance of erythrocytes and the skin for petechiae. These herald a drop in prothrombin activity to 15 % or less. If they appear stop Dicumarol and give Hesperidin methyl chalcone and vitamin K, 60 to 72 mg intramuscularly or intravenously every 3 to 4 hours. Any grade of hemorrhage warrants the use

of intravenous transfusion of *fresh* citrated blood. This blood must be freshly drawn for the prothrombin content drops to ineffectual levels in 24 to 36 hours.

Depo Heparin or Dicumarol should be continued for 4 to 6 weeks.

### The Treatment of Acute Arterial Obstruction

Ischemic gangrene is imminent whenever embolism or thrombosis suddenly obstruct a major or secondary arterial pathway. Occlusion may occur after coronary thrombosis with transmural myocardial infarction, thrombophlebitis *semitis* or diabetic arterial atherosclerosis, or thromboangitis obliterans. The affected extremity becomes cold, pale and cyanotic, the patient feels numbness, burning, tingling, cramps, severe and excruciating pain.

Embolism is an indication for immediate intravenous papaverine hydrochloride injection in doses of 0.1 to 0.3 Gm.  $1\frac{1}{2}$  to  $2\frac{1}{2}$  gr. intravenously first resorted to.

Robert Katz has recommended the use of intravenous or intra-arterial injections of 2.5 % ether in 1000 cc of saline or 5 % glucose daily with a 20 gauge needle attached to a two way adapter. M/6 sodium lactate solution has been given for the same purpose. Buffered sodium citrate solution 2 % has been used and normal and hypertonic saline solutions or 0.02 Gm. % niacin in 5 % glucose or 1 % procaine solution have been advocated as intravenous vasodilators.

Idor Mufson introduced intra-arterial histamine infusion through a 20 gauge needle to counteract obliterative disease of the peripheral arteries. A 500 cc infusion burette is fitted with a clamped in rubber stopper carrying therein a Y tube for attaching a bulb and a manometer. To 500 cc of normal saline solution add 1.38 to 2.75 mg histamine acid phosphate equivalent to 0.5 to 1.0 mg histamine base.

This histamine solution is forced into the artery at the rate of 2 to 5 drops per arterial beat. It produces erythema, flush of the face should be repeated bi-weekly or weekly and injected from a 20 gauge needle.

A 1 % procaine solution has been used for similar purposes, radioactive sodium isotope Na<sup>24</sup> has been used as a tracer substance.

Embolism to the pulmonary artery reproducing the clinical picture of acute cor pulmonale calls for similar emergency treatment along with oxygen under pressure. If the patient fails to rally, pulmonary embolism by a modified Trendelenburg operation should be undertaken if a competent thoracic surgeon is available.

### Status Asthmaticus

The epinephrine fast asthmatic patient develops an acute vesicular pulmonary emphysema that may produce a fatal anoxic state. Emergency measures are often necessary to save the life of the patient who has developed status asthmaticus. Epinephrine, ephedrine, Isuprel, and such sympathomimetic drugs are no longer of help and may do harm. Morphine and its homologues depress the cough reflex and the respiratory center and are thus dangerous and must not be administered. Sedation with barbiturates is not without danger. ACTH is awaited with much hope in this as in other conditions.

**Aminophylline**, 0.5 Gm ( $7\frac{1}{2}$  gr) intravenously was first shown in our clinic in 1931 to be the most effective bronchial dilating agent. Usually the half dose 0.25 Gm ( $3\frac{3}{4}$  gr) in 10 cc ampule is given slowly every 4 hours. A rare patient becomes aminophylline fast. More patients after aminophylline lose the adrenal fastness and respond again to the injections of Adrenalin.

**Oxygen** delivered through an aviator's mask, 6 liters per minute under 2 to 4 cm water pressure, may be necessary at times but usually a tent or even a nasal catheter may supply adequate oxygen. The dilution of oxygen 20 % in helium 80 % will often relieve the patient of much respiratory effort.

**Fluids** as 5 % glucose or 0.9 % saline intravenously may be needed to relieve dehydration. Hypertonic solutions may aggravate matters.

**Passive pressure elevation** of the diaphragm is performed by placing the hands on the upper abdomen under the rib margin and pressing the epigastrium inward and upward during the latter half of expiration augmenting this. Elevating of the diaphragm helps to overcome the acute emphysema by forcing out the trapped air. This should be repeated every few hours until tightness is relieved.

**Bronchoscopy** may be necessary to prevent asphyxia by removing inspissated plugs of mucus and exudate from the bronchus. Antihistaminic drugs favor the formation of inspissated plugs and should therefore be withheld.

**Expectorants** are rather slow in acting and do little in the emergency for they are usually given orally. Sodium iodide 1 Gm (15 gr per 10 cc) may be given slowly intravenously. Potassium iodide 1 Gm (15 drops sat sol) may be administered four times daily by mouth.

**Antibiotics**, aerosol penicillin 50,000 units per cubic centimeter, with or without antihistamines may be sprayed into the bronchial tree by oxygen or air. Intramuscular injections of 300,000 of crystalline procaine

penicillin daily are helpful in overcoming bronchial infection which is often a large factor especially in asthmatic bronchitis

Sedation may occasionally be necessary to allay extreme anxiety. If so Demerol 50 mg may be given as it may have some dilating quality and does not affect the cough and respiratory reflexes as much. It may be repeated in 6 to 8 hours.

### Massive Gastric Hemorrhage

The patient should remain in bed at absolute rest during the time of hemorrhage and until the bleeding has stopped. This is determined by the color of the stools that is when the stools change to a light brown from a tarry black color. If necessary sedation with bromides morphine or the barbiturates may be used to secure complete rest.

When nausea and hematemesis is severe gastric lavage with a weak solution of sodium bicarbonate may be employed through a nasal catheter. The nasal catheter may be left in to relieve any distention present.

Since the quicker the ulcer heals the sooner the hemorrhage will stop diet is important. (See under Diets.) Start the patient on the Andresen Menlengracht or Sippy diet given at 2 hour intervals immediately. Proteolysates as Amigen Caseo Parenamine or Pssename may be given by mouth or parenterally by vein in lieu of foods. Alternate the feedings with 20 to 30 gr of sodium carbonate in water or aluminum gels. Tap water may be given as desired. Vitamin C is given parenterally until fruit juices are added to the diet. Broth Jello and soft foods may be added on the third to the sixth day. A bland diet is started the sixth day if nausea and vomiting do not occur. Alkalies are given after the main meals and at bedtime.

If there has been no bowel movement by the fourth day an oil enema followed a few hours later by a saline enema may be given.

After the hemorrhage has ceased correct the anemia with iron (ferrous sulfate) up to 2 Gm per day.

If the hemorrhage tends to recur surgery consultation is indicated.

Perforations usually call for surgical intervention but gastric suction continuously and antibiotics to control the peritonitis have been successful.

### Acute Dilatation of the Stomach

Rapid distention of the stomach with air may create an extremely serious state and may produce irreversible circulatory collapse or death if it goes on for any length of time unrecognized. It is usually the result of swallowing air which occurs frequently during a comatose state when the patient is gasping or breathing stertorously as a result of

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Continue regime until the patient is urinating freely. Then start a subcutaneous elysis of Darrow's K lactate ( $KHCO_3$ ) 60 to 100 c c per kilogram of body weight.

Continue intravenous saline until moderate degree of hydration occurs, then start oral feedings through a plastic tube.

During the second 24 hours, supplement the intravenous and hypodermoclyses with oral feedings of skimmed milk with lactose or glucose.

Oral Darrow's K solution  $\frac{1}{3}$  to  $\frac{1}{4}$

Glucose 5 % in water  $\frac{2}{3}$  to  $\frac{3}{4}$

Continue giving the patient 200 to 300 c c per kilogram of body weight of total fluids including 5 % Karo corn syrup and a total of 1 to 2 Gm (15 to 30 gr) of NaCl (table salt) and 33 to 65 mg ( $\frac{1}{2}$  to 1 gr) of KCl (potassium salt).

Darrow's solution to be used subcutaneously only. It is made up in distilled water.

$K_2HCO_3$  0.410 Gm KCl 0.210 Gm and NaCl 0.400 Gm in  $H_2O$  100 c c

### Emergency Treatment in Thyroid Crises

Absolute rest in bed in hyperthyroidism is essential and heavy sedation may be necessary. Lugol's solution 10 minims is given every 4 to 6 hours. Glucose and fluid intake must be maintained at a high level. In an acute thyroid crisis Lugol's solution 30 to 60 minims is added to 300 to 600 c c of 25 % or 50 % glucose and given intravenously. Morphine is given unless the patient is sensitive to it and vomits, then chloral hydrate in 0.3 Gm (5 gr) may be given every 3 hours. Propyl thiouracil may be given but it is slow to take effect. If there is hypertension in the crises Benzodioxane might be used.

### Emergency Treatment of a Crisis in Addison's Disease

Give at once intravenously the aqueous *adrenal cortical extract* 20 c c by slow drip 100 c c subcutaneously and intramuscularly 5 c c of the adrenal cortical extract in oil. Repeat in half this dosage every 2 to 4 hours alternating extracts until definite improvement is noted, then stop.

Start immediately a continuous intravenous drip of 5 % glucose in normal (0.9 % NaCl) saline solution 2 c c per minute 100 c c per hour and not more than 2500 c c per day. Discontinue when fluids by mouth are tolerated.

Desoxycorticosterone acetate 5 to 10 mg in oil is injected intramuscularly at once and repeated in 12 hours and then 5 mg daily unless the blood pressure rises and the blood volume is elevated to the point of increasing the size of the heart. After the crisis 1 to 2 mg per day.



metabolic respiratory disturbance or of a central nervous system disorder, or of a very severe infectious disease such as pneumonia, or often after operations. If the patient is conscious, he may complain of a feeling of fullness, nausea, or anorexia, he vomits small amounts of liquid material without getting any relief. He attempts to belch but is unable to do so.

This condition is much less common since the introduction of more or less routine postoperative gastric and upper intestinal decompression by use of the Wangenstein suction apparatus and the Miller Abbott tube. Treatment by gradual decompression by aspirating the air out of the distended viscus and lavaging it results in prompt and complete relief. The contents of the air or fluid should be removed slowly and the stomach lavaged with cool water until the muscle tone returns and peristaltic movements are reestablished. There should be no hesitancy in inserting a stomach tube at the slightest provocation or suggestion of the presence of this serious complication. Removal of the air usually stops the vomiting and forestalls the development of this circulatory peripheral failure.

### The Treatment of Acute Gastroenteritis With Diarrhea

At the onset of nausea, vomiting, and diarrhea give the patient, adult or child the oral buffer solution of sodium lactate (Alexis F Hartmann) made up of lactic acid USP 15 c c sodium hydroxide 10 % sol 20 c c, water up to 100 c c. The dose is 5 c c diluted ten times with water fruit juice or tomato juice, given as often as hourly until the vomiting and diarrhea have been brought under control.

In diarrhea in children, stop food by mouth or limit it to liquids and try to determine the cause and the grade of dehydration and the electrolyte disturbance. Draw blood for sodium potassium chlorides and  $\text{CO}_2$  combining power. If the diarrhea is severe, give fluids to total 200 to 300 c c per kilogram of body weight.

Supply an equal volume of 20 c c per kilogram of body weight of whole blood, plasma, or normal saline.

Give a continuous drip of 5 % glucose in distilled water 100 to 150 c c per kilogram of body weight.

In persistent acidosis with  $\text{CO}_2$  combining power below 15 vol %, administer corrective doses of sodium bicarbonate or M/6 sodium lactate solution.

$\text{NaHCO}_3$  needed =  $\frac{\text{body weight (kg)}}{33} \times 10$  to raise  $\text{CO}_2$  combining power 10 vol %  
 0.2 of the body weight in kg =  $(25 - \text{CO}_2)$  = a millimol  $\text{NaHCO}_3$  or 1 c c M/1 Na Lactate

and autoclave the entire connected apparatus for 15 minutes at a steam pressure of just less than 20 pounds

Cool carefully and then connect the drying tube with a carbon dioxide tank. Bubble the gas through until the pink alkaline solution just becomes colorless indicating a pH of between 8 and 9. This procedure carried to this end point converts the 2.5 % carbonate solution to a 4 % bicarbonate solution. The absorption tube is then carefully removed and the sterile gauze plug replaced.

#### **Intravenous Sugar or Glucose Solution 50 % Stock, 5 % Emergency**

Dissolve 500 Gm. of chemically pure glucose in 1,000 c.c. of distilled water. Solution may be aided by heat. Filter through cloth, cotton and finally paper to remove all of the flocculent precipitate. This stock solution may then be placed in a clean flask, stoppered and autoclaved for 15 minutes at 20 pounds of steam pressure. Dilutions of the stock solution are however usually made before the autoclaving. Transfer 100 c.c. of the stock 50 % solution to a little flask, make up to a 1,000 c.c. with distilled water, plug and autoclave as above in preparing the routine emergency 5 % glucose solution.

#### **Sodium Citrate 2 % Solution**

##### **Anticoagulant for Transfusion Blood**

Dissolve 10 Gm. of chemically pure sodium citrate crystals in 500 c.c. of freshly distilled water. This 2 % solution of sodium citrate is usually perfectly clear; any sediment should be removed by filtering through paper.

The filtered solution may be transferred in smaller volumes to smaller flasks or the stock flask itself may be retained. Plug with gauze covered cotton plugs and autoclave for 15 minutes at 20 pounds of steam pressure.

Allow 10 c.c. of this solution for every 100 c.c. of blood making a 0.2 % concentration in the blood which is sufficient to prevent coagulation and still not a toxic amount.

#### **Sodium Citrate Buffered 2 % Solution**

Warm 250 c.c. and inject daily or every other day according to severity of the symptoms of thromboangitis obliterans.

Use chemically clean glassware.

1. Dissolve 80 Gm. of sodium citrate C.P. and 12 Gm. of sodium chloride C.P. in 4 L. of freshly glass distilled water.

2. Allow to stand for 30 minutes.

is sufficient for maintenance. Counteract shock with warm blankets, oxygen, and transfusions, but avoid all opiates or derivatives and substitutes. If fever or signs of infection develop, use antibiotics or antibacterial agents as penicillin or sulfadiazine.

Reference Sorkin, S. Z. Addison's Disease, *Medicine* 28: 371, 1949.

### Intravenous Therapeutic Solutions

The introduction of solutions directly into the venous circulation is common practice, especially in some of the most urgent emergencies. The sterile towels, gauze, tubing, glassware, including glass ground syringes, and the solutions ordinarily used should be kept in readiness in the emergency ward. Aseptic surgical technique should be used in injecting solutions directly into the blood stream.

The preparation of solutions for intravenous infusion is quite important and requires training, skill, and care.

#### Normal Saline or Physiological Salt Solution 0.85 %

Place exactly 8.5 Gm. of chemically pure sodium chloride in a 1,000 c.c. flask and add exactly 1 liter of freshly distilled water.

Filter the solution through paper into a clean flask.

Plug with gauze covered cotton plugs.

Autoclave for 20 minutes at 20 pounds of steam pressure.

#### Alkali or Sodium Bicarbonate Solution 4 %

If alkalis are to be administered in diabetic coma, it is safer to give small doses and not more than 30 Gm. within a period of 24 hours.

Sodium bicarbonate for intravenous use may be prepared by removing the alkali from a previously unopened container with a sterile spatula and weighing it in a sterile balance pan. The correct amount is dissolved in sterile normal saline solution in a sterile beaker and diluted up to the required amount to make a 4 % solution. This solution does not require further sterilization and should not be boiled because the irritating sodium carbonate will precipitate. CO<sub>2</sub> gas introduced aseptically will change the carbonate back to its original form.

Another method of preparation is to place 25 Gm. of chemically pure anhydrous sodium carbonate in 1,000 c.c. of freshly distilled water. The 2.5 % sodium carbonate solution is filtered through paper and about 5 drops of 1 % alcoholic solution of phenolphthalein indicator.

Immerse an absorption tube (Folin) in the solution in the flask. With rubber tubing connect this to a calcium chloride drying tube filled with absorbent cotton. Plug the flask with gauze covered cotton plugs.

and autoclave the entire connected apparatus for 15 minutes at a steam pressure of just less than 20 pounds

Cool carefully and then connect the drying tube with a carbon dioxide tank. Bubble the gas through until the pink alkaline solution just becomes colorless indicating a pH of between 8 and 9. This procedure carried to this end point converts the 2.5 % carbonate solution to a 4 % bicarbonate solution. The absorption tube is then carefully removed and the sterile gauze plug replaced.

### **Intravenous Sugar or Glucose Solution 50 % Stock, 5 % Emergency**

Dissolve 500 Gm. of chemically pure glucose in 1 000 c.c. of distilled water. Solution may be aided by heat. Filter through cloth, cotton and finally paper to remove all of the flocculent precipitate. This stock solution may then be placed in a clean flask stoppered and autoclaved for 15 minutes at 20 pounds of steam pressure. Dilutions of the stock solution are however usually made before the autoclaving. Transfer 100 c.c. of the stock 50 % solution to a little flask, make up to a 1 000 c.c. with distilled water, plug and autoclave as above in preparing the routine emergency 5 % glucose solution.

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The filtered solution may be transferred in smaller volumes to smaller flasks or the stock flask itself may be returned. Plug with gauze covered cotton plugs and autoclave for 15 minutes at 20 pounds of steam pressure.

Allow 10 c.c. of this solution for every 100 c.c. of blood making a 0.2 % concentration in the blood which is sufficient to prevent coagulation and still not a toxic amount.

### **Sodium Citrate Buffered 2 % Solution**

Warm 250 c.c. and inject daily or every other day according to severity of the symptoms of thromboangitis obliterans.

Use chemically clean glassware.

1. Dissolve 80 Gm. of sodium citrate C.P. and 12 Gm. of sodium chloride C.P. in 4 L. of freshly glass distilled water.

2. Allow to stand for 30 minutes.

3 Test the pH If pH lies between 7.2 and 7.6, proceed If not, adjust by adding a tip of a teaspoonful of potassium diphosphate CP Mix thoroughly and let stand for 30 minutes Read pH and repeat adjustment if necessary

4 Filter through hardened No. 575 Schleicher & Schull filter paper, with metal filter cone and suction flask, a Berkefeld filter

5 Pour into liter flasks and autoclave at 15 pounds for 20 minutes

6 The solution in each bottle must be free of "floaters" or cloudiness, and if not, filter again and autoclave again

The careful suction filtration and refiltration is necessary in order to remove all floaters and thus avoid reactions

Storage in liter flasks covered with glass caps fitting closely over the long necks of the flasks is satisfactory and the autoclaved solution may be kept thusly for months

Flasks should be kept separately and used for citrate solution only Wash with soap and warm water and rinse thoroughly with distilled water before using

To prepare rubber tubing for intravenous therapy, soak new tubing in 5 % NaOH for 24 hours inside and outside Fill the lumen by siphoning its entire length and then immerse it with a weight on it to hold it under Scrub the tubing with soap and water after removing from NaOH solution, and rinse inside and out Boil 3 times for 5 minutes each using fresh water each time, being sure the lumen is full of water Attach to a faucet and allow water to run through it all night

### Vasodilators

#### Solutions for Intravenous Use

**Niacin** (nicotinic acid) Use 100 mg in 500 cc of 5 % glucose Run in just rapidly enough to produce a slight flush

**Histamine acid phosphate** 0.275 mg, equivalent to 0.1 mg histamine base per cubic centimeter ampule is diluted in 250 to 500 cc normal 0.9 % saline or 5 % glucose solution and used for intra arterial injection.

**Ether** (anesthetic quality) 2.5 % in 1,000 cc M/b sodium lactate solution and alcohol (pure ethyl 95 % solution) 5.0 % in 5 % glucose solution have been used as intravenous vasodilators daily for 10 to 12 days

**Alcohol**, 7 % in a 5 % sterile glucose solution is available on the market put up by the Baxter Co and others It is of value intravenously, especially in fat embolism (L. G. Herrmann, M.D., personal communication)

## Protein and Amino Acid Preparations

Parenteral mixtures of amino acids are primarily used to supply dietary nitrogen in readily assimilated form when there is interference with ingestion, digestion, or absorption of food.

Protein hydrolysates should not be employed as a substitute for food proteins if these can be utilized. In severe hepatic insufficiency and in acidosis, intravenous use of hydrolysates is contraindicated.

The oral use of protein hydrolysates should be limited to children allergic to milk, the treatment of peptic ulcer and ulcerative colitis, and in supplementing a high protein diet when this cannot be maintained by ordinary foods.

The ten amino acids considered to be essential at present are phenylalanine, tryptophane, methionine, lysine, leucine, isoleucine, threonine, valine, histidine, and arginine. The exact daily requirements of the individual amino acids are under investigation.

The optimum intake of protein per day is 1 Gm per kilogram of body weight.

There are numerous protein hydrolysates and amino acid preparations on the market for intravenous and for oral use. A few of the better known ones will be discussed.

**Protein Hydrolysate Baxter** (obtainable from Don Baxter Inc., and distributed by American Hospital Supply Corporation)

This parenteral protein hydrolysate is put up in three forms: 5% solution of enzymatically digested protein in distilled water without dextrose; with 5% dextrose; or with 5% dextrose and 7.5% alcohol.

This preparation contains all the amino acids present in bovine plasma proteins and those derived from pancreatic enzyme preparation. Each 100 cc contains 5 Gm of solids, 0.6 Gm of total nitrogen, 0.3 Gm of free alpha amino nitrogen, and 3 Gm of sodium chloride.

Since the normal adult protein requirement is 1 Gm per kilogram or approximately 0.16 Gm of nitrogen per day, 2000 cc of Protein Hydrolysate Baxter should supply the maintenance requirements of a 70 kilogram adult afebrile and with no abnormal fluid loss. To keep a patient in nitrogen balance, the urinary nitrogen excretion for 24 hours should be determined.

**Parenammine** is a product of Winthrop Stearns Inc., New York 13, New York. The full daily adult requirements of protein (60 Gm of amino acids) are contained in one liter of Parenammine 6% solution to be given intravenously.

**Essenammine** is a protein concentrate for oral use put out by Winthrop Stearns Inc. It is bland and unflavored so that it may be com-

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The careful suction filtration and refiltration is necessary in order to remove all floaters and thus avoid reactions

Storage in liter flasks covered with glass caps fitting closely over the long necks of the flasks is satisfactory and the autoclaved solution may be kept thusly for months

Flasks should be kept separately and used for citrate solution only Wash with soap and warm water and rinse thoroughly with distilled water before using

To prepare rubber tubing for intravenous therapy, soak new tubing in 5 % NaOH for 24 hours inside and outside Fill the lumen by siphoning its entire length and then immerse it with a weight on it to hold it under Scrub the tubing with soap and water after removing from NaOH solution, and rinse inside and out Boil 3 times for 5 minutes each, using fresh water each time, being sure the lumen is full of water Attach to a faucet and allow water to run through it all night

### Vasodilators

#### Solutions for Intravenous Use

**Niacin** (nicotinic acid) Use 100 mg in 500 cc of 5 % glucose Run in just rapidly enough to produce a slight flush

**Histamine acid phosphate** 0.275 mg equivalent to 0.1 mg histamine base per cubic centimeter ampule is diluted in 250 to 500 cc normal 0.9 % saline or 5 % glucose solution and used for intra-arterial injection.

**Ether** (anesthetic quality) 2.5 % in 1000 cc M/6 sodium lactate solution and alcohol (pure ethyl 95 % solution) 5.0 % in 5 % glucose solution have been used as intravenous vasodilators daily for 10 to 12 days

**Alcohol**, 7 % in a 5 % sterile glucose solution is available on the market put up by the Baxter Co. and others It is of value intravenously, especially in fat embolism (L. G. Herrmann, M.D., personal communication)

### Dextran as a Plasma Substitute

Various organisms such as *Leuconostoc mesenteroides* cultured in sucrose and phosphate produce dextrans. The protein and inorganic salts are removed and then a solvent similar to acetone precipitates out a polysaccharide composed entirely of glucose units. By means of partial hydrolysis with acid the large molecules of the crude product are reduced to sizes in the plasma protein range. This pale straw yellow liquid has the same specific gravity and saline content as plasma.

Dextran is widely used in Sweden and has recently been introduced into England. It has many advantages over other nonprotein colloids. It will restore blood volume quickly with no toxic pyrogenic or sensitizing effects. It has been found that only whole blood or plasma will maintain blood volume and osmotic pressure as well as dextran. Circulatory overloading must be prevented however particularly in elderly people.



bined with all types of foods to insure an adequate intake of protein and amino acids. It is supplied in 7½ oz bottles. 1 level tablespoon is equal to 8 Gm and is equal to 32 calories.

**Elamine** lyophilized is a hydrolyzed casein with acid tryptophane added and glutamic and aspartic acids partially removed. It contains all the essential amino acids, and no sodium, dextrose, or preservatives. It is supplied in 60 Gm sterile vacuum bottles which may be restored with water, sodium chloride or dextrose sterile solutions to 10 % solution or 800 cc. This 60 Gm bottle satisfies the daily nitrogen requirements of the average surgery patient. This is produced by Interchemical Corporation, Union, New Jersey.

**Protolysate**, or **Protenum** protein hydrolysate for oral use is a product of Mead Johnson and Company, Evansville 21, Indiana. 1 table spoon of Protolysate contains 6 Gm of protein and 30 calories. The average daily dosage can be calculated from this.

**Amigen** (Mead Johnson and Company) is a pancreatic hydrolysate of casein consisting of amino acid and small peptides. It is prepared in sterile nonpyrogenic solution for parenteral administration. The approximate measurements and food values of Amigen are 1 Gm equals 37 calories and is equivalent in nitrogen content to 0.75 Gm of protein, or 1 Gm of protein is supplied by 1⅓ Gm of Amigen.

**Lactamin** (Wyeth Incorporated) is an enzymatic hydrolysate of lactalbumin containing no undigested protein and no insoluble nitrogen, 65 % of which is of amino acids and the balance in polypeptides and peptides. Following is the suggested daily dose schedule.

	PROPHYLAXIS IN SURGERY, TRAUMA, PREGNANCY IN ADEQUATE DIET	SURGICAL OR TRAUMATIC WOUNDS AND BURNS	GASTROINTESTINAL DISEASE AND PROTEIN DEPLETION
Grams per kilogram body weight per day	0.5-1.5	1-5	2-4
Example: a 150 lb patient would receive, in ounces (avoirdupois) Lactamin	1-2-3-6	4-8-12	4-8-9-6

Three level tablespoonfuls of Lactamin equal 1 ounce

**Caminoids** (Arlington Chemical Co. Yonkers N. Y.) is an aminopeptidate from selected protein sources as extracted beef muscle liver, wheat gluten, soya yeast, casein, and lactalbumin. It contains all ten essential amino acids in significant and balanced amounts to ensure optimum utilization with carbohydrates, dextrose maltose, and sucrose to prevent protein diversion. The dose depends upon the individual patient. One level tablespoonful provides 4 Gm of protein as hydrolysate.

in the body Sulfadiazine sulfamerazine and sulfacetimide (Sulamyd) are the three most widely used sulfonamides at present

Sulfadiazine as first choice, and its homologues as second and third choices are recommended for common infections due to beta hemolytic streptococci such as acute tonsillitis quinsy adenitis cellulitis sinusitis otitis media erysipelas and puerperal sepsis The sulfonamides are valuable in treating pneumococcal pneumonia staphylococcal septicaemia, Friedlander's infection actinomycosis tularemia, meningitis (meningococcal staphylococcal streptococcal and influenzal), *E. coli* infections and venereal diseases as chancreoid lymphogranuloma inguinale and gonorrhea which is not resistant to sulfonamides

Since the advent of such antibiotics as penicillin and streptomycin these have been used in combination with the sulfonamides with better results than either one alone

Various mixtures of sulfonamides have been found to be less toxic than a single drug given alone that is the prevention of renal complications is less A double sulfonamide mixture sulfadiazine and sulfamerazine is recommended in a sulfonamide susceptible infection except meningitis

Sulfadiazine sulfaguanidine and better still Sulfasuxidine and Sulfathalidine are extensively used in preoperative preparation of patients with surgical bowel conditions Nisulfazole in ulcerative colitis bacillary dysentery cholera and *E. coli* infections, and sulfacetimide in chancreoid and urinary tract infections

### Antibiotics

Penicillin is the most effective therapeutic agent available for the treatment of many infections due to gram positive organisms and spirochetal infections Penicillin G of the four types of penicillin is the most widely used clinically It is highly effective in staphylococcal infections such as pneumonia meningitis endocarditis osteomyelitis soft tissue abscess wound infections gonorrhea, and mastoiditis It has been found to be useful combined with sulfadiazine or a similar sulfonamide in certain types of infections as streptococcal pneumonia empyema peritonitis meningitis septicaemia mastoiditis or Ludwig's angina It is also useful in meningococcal infections when the sulfonamides are ineffective, or the two drugs may be used together Crystalline penicillin G is the drug of choice in syphilis yaws mal de pinto relapsing fever and actinomycosis

Allergic reactions due to sensitivity to molds may be controlled with antihistamine drugs but in some cases make necessary the use of another drug

## Chapter XXVIII

### GENERAL THERAPEUTIC METHODS

Tremendous advances have been made in the science and in the art of therapy in the half century that has passed since Dr George Dock inaugurated his lectures on "The Necessary Twenty Drugs" at the University of Michigan in 1899. A quarter of a century has elapsed since his stimulating medical service at Washington University closed and this manual was first published. Most of the twenty drugs are still in use but many have been superseded by more active agents. The chemotherapeutic agents, antibacterial agents, antibiotics, antihistaminic drugs, newer sedatives as barbiturates and anesthetics and narcotics have revolutionized medical practice. Organic chemists have analyzed and synthesized hormones and vitamins and many effective agents.

In the treatment of infectious diseases the very effective sulfonamides are used. Better and better sulfur drugs are still being developed, only to be superseded by the more spectacularly acting antibiotics beginning with penicillin, tyrothricin, bacitracin, then streptomycin and its dihydro derivative. Aureomycin followed and greatly extended the field of usefulness and became pre-eminent, only to be challenged by the advent of Chloromycetin and its synthetic form chloramphenicol. The whole field of mycology is being vigorously investigated and soil fungi from every corner of the earth are being tested for the production of antibacterial, antimicrobial, and antiviral substances of even greater potency than those already discovered. Terramycin controls an ever wider spectrum of pathogens.

The antibiotics have removed the sting of death from a great many diseases caused by virulent bacteria, viruses, rickettsia, and fungi. At this time the viruses of poliomyelitis and encephalitis and a few other bacterial and rickettsial and viral diseases have as yet not been conquered. Tuberculosis, rabies, coccidioidomycosis and histoplasmosis are still somewhat resistant. In fulminating infections which are overwhelming, the antibiotics are used with some justification before the microbiological laboratory has had time to identify the causative organism.

#### Antibacterial Chemotherapy and Antibiotic Therapy

**Sulfonamide Therapy**—Besides knowing the causative organism responsible for the infection and choosing the sulfonamide on this basis it is important to choose the drug according to the location of infection.

kapsal, 3 to 4 times daily for 5 to 7 days usually completes a course  
In ulcerative colitis the course should last at least 30 days

Para-aminobenzoic acid has been found to be of value in attacking a group of diseases the rickettsial infections for which previously there was no effective agent. However, aureomycin and Chloromycetin have been found to be of value against this same group of organisms and may prove to be the best agents. PABA has possibilities in the treatment of lymphoblastoma cutis certain forms of lupus erythematosus, dermatomyositis scleroderma and dermatitis herpetiformis. Para aminobenzoic acid is administered in 1 Gm. doses every 4 hours (Brewer and Company, Inc.)

Nisulfazole has been found by Majors to be most effective in ulcerative colitis but Chloromycetin is more promising

**Aureomycin Therapy**—Aureomycin which is produced by *Streptomyces aureofaciens* has been found to be useful in the treatment of a large variety of infections. The rickettsial diseases Rocky Mountain spotted fever typhus fever Q fever and Brill's disease have been treated successfully with aureomycin. It has been particularly effective in Q fever patients. Aureomycin is the treatment of choice in lymphopathia venereum and lymphogranuloma inguinale. There have not been enough controlled results reported as yet in psittacosis and pericarditis. It has a marked beneficial result in cases of primary atypical pneumonia as well as in many of the bacterial pneumonias pneumococcal staphylococcal and Hemophilus influenzal infections of the lungs. Aureomycin has been found of value in Salmonella infections and many staphylococcal infections of the newborn infant skin infections complicating leukemia and osteomyelitis. Many urinary tract infections except those due to proteus and pyocyaneus are treated with aureomycin. In combination with streptomycin it may eliminate urinary tract infection when all other treatment has failed. Aureomycin has been found to be useful in the treatment of acute brucellosis acute peritonitis various eye infections as corneal ulcers follicular conjunctivitis trachoma and keratitis tularemia and lymphocytic choriomeningitis. Dosage 0.25 to 0.50 Gm. by mouth every 4 hours but dosage must be reduced if nausea affects the patient.

**Terramycin** is a crystalline antibiotic isolated from both cultures of *Streptomyces rimosus*. This antibiotic has shown the widest spectrum of antibacterial activity in vitro and in vivo against pneumo strepto-, gono- and staphylococci bacillary infections as anthrax *E. coli* *A. aerogenes* *B. abortus* *B. melitensis* *B. suis* *H. pertussis* *T. pallidum* syphilis *D. granuloma inguinale* virus lymphopathia venereum pneumonia herpes zoster *R.* of epidemic murine or scrub typhus, and *E. histolytica* amebiasis

Penicillin aerosols from nebulizers of mist of 0.3 to 3 microns in diameter or dust particles in doses of 50,000 to 100,000 units in 1 or 2 cc of water, every three hours, are most effective. Dosage varies in procaine crystalline penicillin G, 100,000 units every 3 hours in water, or in peanut oil and beeswax, 600,000 units (2 cc) every 8 to 12 hours.

**Streptomycin** is indicated in the treatment of infections due to susceptible gram negative organisms. It is recommended in the treatment of tularemia, pertussis, Hemophilus influenza meningitis, endocarditis pulmonary tract infections, and urinary tract infections in bacteremia due to *E. coli*, *K. pneumoniae*, *B. pyocyaneus*, *P. vulgaris*, and *A. aerogenes*, as well as urinary tract infections due to similar gram negative organisms. It has been found to be a valuable adjunct in the treatment of tuberculosis various types of endocarditis chronic pulmonary infections due to gram negative flora and streptomycin sensitive organisms and in empyema and various types of peritonitis due to gram negative organisms as well as lympho granuloma inguinale.

Streptomycin aerosols are given in respiratory infections especially pertussis, from nebulizers producing 2 to 5 micron droplets in dosages of 25,000 to 50,000 units every three hours. Little systemic absorption or intoxication takes place. Streptomycin or dihydrostreptomycin 0.5 to 1.0 Gm daily or bidaily may be given but it must be stopped when 8th nerve symptoms appear. Eighth nerve damage has been less since the dihydro form of streptomycin has been developed by Merck & Company, Inc.

The combined use of streptomycin and sulfadiazine has proved to be of value in the treatment of acute brucellosis and H. influenza meningitis.

**Chloromycetin** or chloramphenicol (Parke Davis & Company) has been obtained from the Caracas mold *Streptomyces venezuelae*, but has also been prepared synthetically. It is effective against a wide range of microorganisms. Enteric infections such as typhoid fever and other Salmonella infections, bacillary urinary infections and certain rickettsial diseases as scrub typhus typhus fever, and Rocky Mountain spotted fever all respond to Chloromycetin therapy. Patients with active undulant fever have responded favorably to treatment with Chloromycetin. Although of value in chronic brucellosis the administration of the drug must be prolonged. The use of Chloromycetin is still in the experimental stages in cases of atypical pneumonia, psittacosis lymphocytic choriomeningitis, lymphogranuloma inguinale and colitis.

Chloromycetin is administered orally with daily doses of 2 to 3 Gm 8 to 12 capsules, divided into 2 to 4 doses. After 3 or 4 days, 0.25 Gm 1

kapsal, 3 to 4 times daily for 5 to 7 days, usually completes a course. In ulcerative colitis the course should last at least 30 days.

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**Tyrothricin** (gramacidin and tyrocidin) is an alcohol soluble, water insoluble derivative of an autolyzed culture of aerobic sporulating soil, *Bacillus brevis*. This was the pioneer antibacterial substance but its toxicity was such that its use has been confined to topical application on the surface in ulcers or cavities. Gramacidin is active against gram positive organisms and tyrocidin against gram negative organisms as gonococci and meningococci.

**Bacitracin**—This antibiotic principle was isolated from a strain of *Bacillus subtilis*. Its range of activity parallels that of penicillin; however, fortunately no cross sensitivity to the two antibiotics has been noted.

Bacitracin is used when sensitivity to penicillin is to be avoided. It is useful in the form of ointments or solution in the treatment of open wounds of the eye and in superficial infections of the skin as furuncles, impetigo, or other dermatoses such as allergic eczema, scabies, or contact dermatitis.

**Neomycin**—Neomycin, an antibiotic produced by another of the genus *Streptomyces*, *St. fradiae*, is active against numerous gram positive and gram negative organisms, particularly the mycobacteria, but not against the fungi. It has been found to be active against streptomycin sensitive and streptomycin resistant bacteria and to show particular action against various strains of mycobacterium tuberculosis. Work in the future will indicate its further usefulness clinically. **Polymyxin D** is toxic.

Antibiotics, by suppressing normal bacterial flora in the gut, may contribute to avitaminosis which must be watched for and treated with vitamin concentrates.

### General Management in Acute Infectious Diseases

The patient with an acute infectious disease is strictly confined to bed. This is the first and most important measure because of the acute toxemia which might cause much organ damage, Zenker's necrosis, in most of which fortunately this pathology is of a temporary nature. The patient's water balance and nutrition should be maintained. The fluids should be forced to at least three liters per day. Nausea, vomiting or diarrhea should be treated with oral Hartmann's solution of sodium lactate. This may be given by mouth every hour. The oral buffer is made up of lactic acid USP, 15 c c, 10 % sodium hydroxide 20 c c, and water up to 100 c c. This solution diluted 10 times is to be taken as often as once every hour. A liquid or at least a soft diet of palatable easily digestible nutritious food should be served. A gentle laxative or a mild natural laxative as Mucilose is to be given in small doses with the soft diet to insure bowel function by natural stimulation for elimination and to pre-

vent distention Cathartics should be avoided Fresh air and sunshine and reassurance and relaxation are important adjuncts to therapy

Symptomatic treatment should be administered according to the indications and the patient's general condition Good nursing care is still most essential He may be given tepid  $70^{\circ}$  to  $85^{\circ}$  F ( $16^{\circ}$  to  $24^{\circ}$  C) tap water enema or, if necessary, cool sorapuds may be substituted to help in evacuation Sometimes colonic irrigation helps to reduce high temperatures Irrigation will often help to remove tympanites A sponge bath  $80^{\circ}$  F ( $30^{\circ}$  C) or more is usually a good tonic

The patient must be carefully watched for complications a rise or fall in temperature circulatory failure pulmonary edema gastric dilatation delirium in pneumonia cyanosis or shock Oxygen therapy should be continuous over long periods of time A rise in pulse and drop in blood pressure suggest hemorrhage and call for transfusion A sharp pain in the abdomen may be due to viscus perforation and peritonitis This should be surgically drained if necessary and intraperitoneal intravenous and oral antibiotics may be used

In epidemics such as influenza the secondary invaders pneumococci and streptococci may be controlled In general under such conditions epidemics may be controlled in much shorter time Strict isolation technique need be maintained for no longer than a brief period because the technique interferes with the adequate treatment of the patient professional services costly materials and personnel necessary to maintain strict isolation

## Management of Infectious Diseases

### ISOLATION TECHNIQUE

The treatment and management of contagious diseases in spite of the advent of powerful sulfonamides and antibiotics still calls for extreme care in the handling of patients with fulminating infectious diseases The physician must protect himself other patients and the public Patients admitted with a high fever should be suspected of having contagious diseases which may be transmitted directly from host to host Strict isolation should be invoked until the diagnosis has been established and in certain cases maintained permitting no visitors Mards orderlies nurses and physicians entering the sickroom or cubicle should put on a gown and mask Only authorized instructed persons should be allowed to see the patient

The gown the inside of which is uncontaminated should hang on a hall tree in the sickroom A pair of gloves should remain in a basin containing an antiseptic solution for the hands as cresol 0.5 % solution or



mercury bichloride 1 2,000 solution The gown and rubber gloves should be worn while caring for the patient

Articles necessary for the patient's toilet must be left in the room Towels and all linens are to be removed from the sickroom and put directly into the sterilizer All things pertaining to the patient are to be considered contaminated, and sputum, exudates, urine, and feces are to be placed in the sterilizer, the top closed down with the foot lever and the attendant continues the autoclaving for 20 minutes

The exchange of supplies or materials between patients is to be strictly forbidden

Specimens for laboratory studies are to be placed in externally un-contaminated receptacles or containers and stoppered and labeled "contagious" The stool and urine, after bacteriological and chemical examination, are to be mixed with equal parts of milk of lime, a solution of quick lime (calcium oxide made from freshly slacked lime and water in the proportion of 4 to 1) and allowed to stand in covered basins for one hour Before leaving the sickroom, the attendant should remove the gloves in the disinfecting solution leave them there remove the gown with great care so as not to contaminate it on the inside, and hang it on the hall tree

After leaving the patient's room, the physician puts his contaminated instruments and material into the sterilizer Hands and arms are scrubbed thoroughly with pHIsoderm or green soap After scrubbing the physician should turn on the steam sterilizer, in which he has placed his wrapped instruments and gloves and continue sterilization under pressure for 20 minutes The sterilizers are to be emptied as soon as possible after sterilization has been completed

### **The Care of Infectious Disease Cases**

In infectious diseases involving the skin, care must be taken to de-contaminate whatever the patient touches The thermometer, hot water bottle tileum and bath lotion should not be removed Furniture in the room should be draped with sheets Thin towels should be placed around the patient's chest and arms or wherever the bags of the blood pressure apparatus are applied A contaminated stethoscope should be dipped in the 1 200 bichloride of mercury or 0.5 % cresol solution

The patient's bath water as well as his excreta, should be disinfected with cresol or quick lime Care should be taken that there are no vermin in the sickroom or any intermediate object which might serve to transmit the infected agent from host to host

In acute respiratory diseases of all types acute pharyngitis bronchitis pneumonia, lobar pneumonia or influenza patients as well as the

attendants, should wear masks to cover the nose and throat. They should use Kleenex and expectorate or sneeze or cough into it. A paper bag is attached to the bedclothes at the edge with a safety pin to catch the tissues. When the bag is about half full it is burned.

In gastro intestinal infections as cholera dysentery, salmonella and typhoid fever all excreta should be sterilized in cresol solution as all bowel movement urine or expectoration.

Isolation technique is best maintained in a room or a small ward, but if necessary in a larger ward part of which is screened off into cubicles. It is preferable to use the cubicle sheet system and to provide paraffined paper receptacles for soiled napkins vomitus and excreta. An antiseptic solution is used not only for the hands of the attendant and the patient but also for the expectoration urine and feces.

The patient should be taught to cover his nose and mouth when he is sneezing or coughing and when he is being examined or quizzed he should be equipped with a gauze mask or guard held in place by an attendant.

The length of quarantine has been greatly shortened with the help of newer antibiotics and therapeutic agents which dramatically abort the period of invasion.

In poliomyelitis for instance the isolation period has been placed at one week beyond that it is difficult to provide the necessary personnel to control the disease. The mode of transmission and character of the carriers are still unknown.

### Oxygen Therapy

Anoxia whatever be its cause or the accompanying disease processes is most effectively and successfully treated by administrations of pure oxygen promptly used in an adequate concentration. The patient should be reassured that oxygen is merely given to facilitate his recovery. Fears and anxieties due to the layman's conception that oxygen is used only as a last resort should be dispelled. This is best done by psychological preparation of the patient by the physician and an intelligent nurse before the apparatus is brought into the room. Confidence, calmness and high nursing skill are necessary for the maximum results. A constant supply of oxygen in large 244 cubic foot cylinders should be available so that no interruption in the administration is necessary.

Regulators complete with connections gauges for cylinder pressure or content small adjusting valves meter flow gauges are necessary equipment to reduce high oxygen pressure safely and efficiently and to control the rate of flow to the patient. Some suggestions as to the operation of the equipment and some notes as to the principles and objectives

of therapy are in order. The *Oxygen Therapy Handbook* by the Linde Air Products Company, New York, should be used as a guide. The choice of the equipment for the administration of oxygen depends, of course, upon the devices that are available and the concentration desired. If an atmosphere of only 40 to 50 %  $O_2$  concentration is prescribed, catheter, mask, or tent will be satisfactory. If more than 60 % oxygen concentration is desired, special oronasal aviators' BLB or OEM or Puritan masks are necessary and must be selected according to the indications.

The oxygen cylinder should be "cracked" by opening it slightly and closing it quickly to blow out any accumulated dust. Before handling the oxygen regulator and placing it by the oxygen cylinder, be sure that it is clean and fresh and free of oil droplets, and not previously used for  $CO$  or helium. No grease or oil should be anywhere in contact with  $CO_2$ . If the regulator has been used for  $CO_2$  or helium or either gas with oxygen, it must not be used for pure oxygen without expert cleaning. With  $CO_2$  or helium, compression of the lubricating system may have caused fine particles of oil to become lodged in the regulator and these are likely to explode in the presence of pure oxygen under pressure. If the regulator is contaminated, send it back to the manufacturer for cleaning.

Be sure that the flow adjusting valve is closed, turned to *off*, which is to the left, until it is loose. The cylinder should be opened slowly. Turn the adjusting screw to *on*, which is on the right, until the oxygen flows through the regulator at the desired rate. If the regulator is not used, the flow adjustment screw is turned to the left as far as it will go, to the position marked *off*. In use check the regulator periodically for leaks and liter flow accuracy to prevent waste.

The patient's confidence and cooperation should be gained before the mask is applied. The mask is applied to fit snugly but not tightly. The flow control of the oxygen is started at 6 to 8 liters per minute, even 10 to 12 if necessary. After the patient has completed the first deep inhalations satisfactorily, then reduce the flow of oxygen so that the breathing bag almost but does not quite collapse during inhalation.

Remove the mask every two hours while the patient's face is dried. His hair is moistened and drinking water and food are administered. The hose from the mask to the meter is connected again to the nipple distal to the meter. This water column or manometer should fall and rise sharply as the patient breathes. The mask is attached by a tube directly to the distal nipple on the meter and the proper flow is corrected to the desired rate.

The breathing depth and rate is influenced by the action of the bag which fills with expiration and empties with inspiration. The bag should

not collapse completely during regular normal breathing. If it does the liter flow should be raised and the liter disk reset.

The plug in front of the OEM and Puritan masks may be taken out and a suction tube inserted to remove the saliva and secretions. These masks are equipped with specially designed large diameter tubing which should always be used. Care should be taken that the tubing is not pinched under the patient's arm.

**Concentration of Oxygen Administered**—Concentrations of almost 100 % may be obtained by adjusting the liter flow so that the breathing bag never collapses completely during regular breathing. Oxygen is supplied at the rate of 6 to 8 liters for a small person and 8 to 10 liters for a larger person. Concentrations of 40 to 50 % oxygen may be obtained and the rebreathing bag of the BLB mask collapses with each inspiration on the average patient with a flow of 4 liters per minute. However 60 to 80% is obtained by a flow of 5 to 6 liters per minute. The reservoir bag must not collapse when the high concentrations of oxygen are desired during inspiration. The longer the bag stays collapsed the lower will be the oxygen concentration in the inspired air.

Certain clinical conditions such as acute pulmonary edema require oxygen under pressure. The positive pressure apparatus requires the presence of a physician or specially trained technician. Only slight positive pressures are prescribed. Positive pressure type of OEM or Puritan meter masks have the rubber face piece which will permit administration of oxygen under pressure equivalent to 1, 2, 3 or 4 cm. of water. The resistance is the same as that experienced in exhaling through a tube immersed in water to the same depth. The largest hole in the disk in the mask is used when positive pressure is no longer required. The pressure prescribed is obtained by setting the rotating calibrated disk on the face piece at the hole corresponding to the pressure desired. If the pressure desired is 1 cm. of water pressure it should take 5 or 10 minutes to increase it up to the maximum pressure.

The nasal mask alone, metal nasal inhalers and catheters at routine deep or shallow position are used for lower concentrations.

French catheters sizes 8 to 12 are most convenient. They should be lubricated with water soluble acacia jelly to avoid dryness and placed alternately every 12 hours into the right and then the left nostril to reduce irritation of the nasal mucous membrane. The oxygen is dribbled through distilled water as a humidifier. The distance from the nose to the ear lobe is measured and marked on the catheter by a piece of adhesive or Scotch tape. When the acacia lubricated catheter is inserted into the nostril the oxygen is turned on and should flow through the tube at about 5 liters per minute providing a 30 %  $O_2$  atmosphere. If the

catheter is inserted too far into the pharynx, the patient will swallow repeatedly. If it is not inserted far enough, the oxygen will be wasted and the catheter will come out. Metal tubes with bulbous ends may be fitted into both nostrils and replace the mask or catheter.

When oxygen therapy is discontinued, the mask and all the catheters and tubes should be washed clean of acacia jelly with soap and water and dried thoroughly.

Oxygen by tent is the most pleasant but at the same time the most expensive way of administering oxygen. This is especially true if the cooling of the atmosphere, the humidity, and the concentration of oxygen desired is high. The maximum obtainable in a tent under the best circumstances is 60 %. The atmosphere must be repeatedly analyzed.

The tent is a plastic canopy about 3' x 3' x 3' to enclose the head, neck, arms, and chest to the waist. It must be tucked snugly under the head of the mattress and secured tightly under the covers at the waist. A rubber sheet or plastic sheet is placed over the mattress. The zippered openings or sleeves must be opened as infrequently as possible. Leaks in the tent and in its application must be avoided and the free edges tightly secured all around.

Tents must be equipped with cooling and CO<sub>2</sub> absorbing devices. A motor blower may suck the air of the tent through a tank of soda lime and over ice in a compartment and circulate it back into the tent.

The oxygen flow should be at the rate of 15 liters per minute for half an hour in order to allow a 50 % O<sub>2</sub> atmosphere which may be maintained at 10 liters per minute. The atmosphere is controlled by frequent analyses and changes indicated by the flow rate. A full cylinder of oxygen flowing at 10 liters per minute will last approximately 9 hours, at 8 liters per minute approximately 14 hours, at 6 liters per minute approximately 19 hours, flowing at 4 liters per minute, a standard cylinder of oxygen would last approximately 28 hours. For details see *Oxygen Therapy Handbook*.

### Histamine Antagonists or Antihistaminic Drugs

When histamine was identified as a source of reaction in an allergic condition a search was begun for antihistaminic agents. Torantil was first extracted from the mucosa of the small intestine of the swine but it was found to be too toxic. Organic chemists then synthesized a group of drugs related to the phenolic ester, and ethers and a group of derivatives of ethylene diamine. All of these have the property of diminishing or preventing the effects of histamine without producing the opposite pharmacological reaction. A whole host of these drugs have been developed all having similar effects differing only in side effects and dosage. They are all rather slow in action compared to epinephrine which is still the best drug in allergic crises although ACTH may supersede it.

Antihistamines do not prevent allergic responses in the body and do not prevent the invasion of virus or do anything more than mask the secondary infection. They are palliative treatment for the minor allergy such as hay fever perennial hives vasomotor rhinitis food and drug sensitivities contact dermatitis urticaria pruritus and erythema multiforme. Some benefit is often obtained in patients with bronchial asthma asthmatic bronchitis angioneurotic edema eczema cephalgic, migraine gastro intestinal food allergy and radiation sickness.

The usual oral administration results in consequent absorption delay but is the only satisfactory route for the regular production of systemic effects for the drugs. Doses vary from 25 to 100 mg three or four times a day. The drugs may be used in smaller doses prophylactically. The side effects must be remembered particularly if the patient is driving an automobile and must remain alert and attentive. Drowsiness may be extreme or dizziness or nervousness particularly mental confusion weakness and fainting have been noted. Local administration of the drug is often without general effect and may be successful especially as an aerosol. Ointment aerosols capsules pulvis and elixir (noncarbohyd rate) have been prepared. The intravenous administration is not without danger and should be avoided except in emergency crises.

Beradryl (Parke Davis) the pioneer of this series is diphenhydramine hydrochloride. It is supplied in capsules 25 to 50 mg, in an elixir 0.5% and in 1.09% sterile solution.

Diphenhydramine HCl is the Council adopted name for  $\beta$  dimethyl amino ethyl benzohydry ether hydrochloride.

Pyribenzamine (Ciba) is tripeleminamine hydrochloride and causes less drowsiness but more gastro intestinal side effects. Pyribenzamine is supplied in tablets of 50 mg elixir 0.5% ointment or cream 2%.

catheter is inserted too far into the pharynx, the patient will swallow repeatedly. If it is not inserted far enough, the oxygen will be wasted and the catheter will come out. Metal tubes with bulbous ends may be fitted into both nostrils and replace the mask or catheter.

When oxygen therapy is discontinued, the mask and all the catheters and tubes should be washed clean of acacia jelly with soap and water and dried thoroughly.

Oxygen by tent is the most pleasant but at the same time the most expensive way of administering oxygen. This is especially true if the cooling of the atmosphere, the humidity, and the concentration of oxygen desired is high. The maximum obtainable in a tent under the best circumstances is 60 %. The atmosphere must be repeatedly analyzed.

The tent is a plastic canopy about 3' x 3' x 3' to enclose the head, neck, arms, and chest to the waist. It must be tucked snugly under the head of the mattress and secured tightly under the covers at the waist. A rubber sheet or plastic sheet is placed over the mattress. The zippered openings or sleeves must be opened as infrequently as possible. Leaks in the tent and in its application must be avoided and the free edges tightly secured all around.

Tents must be equipped with cooling and CO<sub>2</sub> absorbing devices. A motor blower may suck the air of the tent through a tank of soda lime and over ice in a compartment and circulate it back into the tent.

The oxygen flow should be at the rate of 15 liters per minute for half an hour in order to allow a 50 % O<sub>2</sub> atmosphere which may be maintained at 10 liters per minute. The atmosphere is controlled by frequent analyses and changes indicated by the flow rate. A full cylinder of oxygen flowing at 10 liters per minute will last approximately 9 hours, at 8 liters per minute approximately 14 hours, at 6 liters per minute approximately 19 hours, flowing at 4 liters per minute a standard cylinder of oxygen would last approximately 28 hours. For details see *Oxygen Therapy Handbook*.

## The Treatment of Blood Dyscrasias

Anemia may be the presenting finding or abnormality in patients with acute and with chronic diseases and in serious situations. It is highly desirable to determine the cause and the type of anemia before any treatment is started or transfusions are given.

Acute massive hemorrhage causes a very serious situation, even to exsanguination. When the red cell count drops below two million and the hemoglobin below 40 % and symptoms of shock appear, it is necessary to replace the blood loss immediately by an injection of whole blood plasma or substitutes.

The patient should be quickly examined, his blood typed and matched and fresh donors should be prepared. The source of bleeding should be discovered and, if possible, this should be stopped. The degree and type of anemia should be established.

Transfusions given carefully and gradually in total amounts of 1000 to 1500 c.c. of fresh whole matched blood are often necessary to bring the blood volume and blood pressure back above critical levels.

In anemia of chronic blood loss in the urine or from hemorrhoids or from the nose or other mucous membranes in epistaxis and in scurvy or purpura the chronic drain may lead to exhaustion of the bone marrow and a hypoplastic or aplastic dyscrasia.

Hemophilia may produce a hypochromic microcytic anemia. In acute hemophilic bleeding the application of cold and injections of histamine 0.1 c.c. 1:1000 solution daily and increased to 1 c.c. and the transfusion of blood or plasma or Cohn's fraction I gamma globulin, if available or the intradermal injection of histamine may be necessary to save a life.

Anemia is a physiologic rather than a morphologic problem. The importance of anoxemia as a marrow stimulant is stressed. Stuppling of the red cells is a form of reticulocytosis. Chlorosis has almost disappeared.

In general such patients should be placed on a diet high in iron and given ferrous sulfate or gluconate pills 0.2 to 0.4 Gm (3 to 6 gr) 3 times a day and conservative therapy should be resorted to unless the condition becomes critical then heroic measures are justifiable.

The fundamental defect in hemophilia is the slow conversion of prothrombin into thrombin. In this disease also the platelets are not supported by proper fibrin formation.

In secondary anemia where the blood hemoglobin has not dropped to critical levels and the cause toxic agent or chronic infection has been stopped blood may be built back by a diet that is high in iron.



Neo Antergan (Merck) is pyramisamine maleate and is supplied in 25 and 50 mg coated tablets

Neohetramine (Nepera Wyeth) is thonzylamine hydrochloride and has produced few side effects. It is supplied in 50 and 100 mg tablets

Decapryn (Merrell) is doxylamine succinate and is soporific and long acting even in 25 mg tablet doses

Trimeton (Schering) is prophepyridamine and is very effective in 25 mg tablet doses

Histadyl (Lilly) Thenylene (Abbott) is thenylpyramine hydrochloride and is supplied in 25 to 50 mg and 100 mg tablets

Antistine (Ciba), phenazoline hydrochloride sulfate, and methane sulfonate is supplied in 100 mg tablets, 1 % elixir, and 0.5 % solution, it may produce insomnia

Thephorin (Hoffmann La Roche) is phenindamine acid tartrate and is potent and stimulating in doses of 25 mg in coated tablets

Chlorothen (Tagathen, Lederle) chlorothen citrate is effective in 25 mg doses in capsule or tablet and 2 % ointment

Diatrin (Warner), N,N dimethyl N' phenyl N' (2 thienylmethyl) ethylene diamine monohydrochloride, rarely produces side effects in 40 mg coated tablet doses

Pyrrolazote (Upjohn), thioldiphenylamine derivative, pyrrolidine ethyl phenothiazine (pyrathizine), an effective antihistamine of low toxicity in 25 to 100 mg tablet doses

Bromothen (Lederle) is similar to Chlorothen but the bromine derivative Brombenzyl DPE (Ciba) is a bromide derivative of tripelenamine pyribenzamine

Hydrylin (Searle), diphenhydramine 25 mg and aminophylline 100 mg, is a potent and effective combination

Dramamine (Searle), dimenhydrinate, in 50 mg doses is particularly effective in the prevention of motion sickness

Perazil (Burroughs Wellcome), chlorcyclizine in 50 mg doses produces minimal side effects

New sympathomimetic bronchodilators

Orthoxine Hydrochloride,  $\alpha$  (ortho methoxyphenyl) isopropyl methylamine HCl (Upjohn) In asthma as prophylaxis, 100 mg, three or four times a day

Isuprel Hydrochloride (Winthrop Stearns) 1 (3' 4' dihydroxyphenyl) 2 isopropylaminoethanol, in tablets 10 mg to 15 mg sublingually and in solution 10 c.c. (5 mg/c.c.) for inhalation

In sickle-cell anemia the spleen is only occasionally found enlarged and its removal is of little avail. Oxygen and transfusion of oxygenated blood usually help. In fatal cases the spleen is shrunken.

**Toxic anemia**—Substances as potassium chlorate, benzol, phenol or naphthol or malaria or syphilis may be producing or be responsible for anemia and if so they should of course be removed or healed if possible.

**Macrocytic Anemia**—Castle proved that macrocytic anemia was a deficiency disease by the biologic assay of gastric juice of P. A. cases. He showed that achylia gastrica played an intermediary role in pernicious anemia with its intrinsic and extrinsic factors. Normal man's stomach contents one hour after 300 Gm. of ground beefsteak treated with dilute HCl, pH 2 to pH 3, was incubated 6 hours, then neutralized with NaOH to pH 7 and given fasting, by tube. This beefsteak was effective 8 out of 10 times giving the characteristic reticulocyte response. Hamburger digested with commercial pepsin and 150 Gm. pig's mucosa incubated as above was ineffective 3 out of the 10 cases and in two others 300 Gm. of hamburger was always ineffective alone or with HCl. Castle concluded that Addisonian pernicious anemia is a deficiency disease conditioned by the lack of a specific intrinsic factor present in the normal human gastric juice and absent in P. A.

In the normal individual the function of this intrinsic factor in the gastric juice is to interact with an extrinsic factor in the food to produce specific hematopoietic effects as a rise in reticulocytes, erythrocytes and hemoglobin in P. A. within ten days.

The intrinsic factor is heat labile, not corresponding to HCl, pepsin, rennin or lipase. The extrinsic factor is in washed beef muscle precipitate at pH 6 in yeast but not in washed casein or wheat gluten.

Vitamin  $P_{12}$  in 10 microgram injections is equivalent to 1 USP unit of liver oil, *vit. mucosa* and produces reticulocytosis developing in the bone marrow in 12 hours and in the peripheral blood in 48 to 72 hours to a maximum in 5 to 7 days. It relieves the gastrointestinal symptoms, soreness and burning in the mouth, the loss of the appetite, general alertness, nutrition and the weight are rapidly increased. The biological manifestations are conspicuous.

The macrocytic anemia of cirrhosis of the liver and the depressed erythropoietic state have not been found to respond to the liver extract in the usual way. It must be remembered that the leucopenia is only moderate but there is usually disturbance in the blood proteins, low serum albumin, elevated serum globulin and lowered A/G ratio. The treatment should be directed against the liver condition by a high pro-

containing foods and high in proteins which are of a good biological character and help in the rebuilding. In addition, ferrous sulfate or gluconate pills, 0.2 to 0.4 Gm (3 to 6 gr) 4 times a day may be added.

### Hypoplastic Anemia

Complications of severe chronic bleeding are drawn on the bone marrow. There may be severe depressions from severe prolonged infection. If the patient has been taking any drugs as sulfonamides, arsenicals, gold, salts, thiouracil, imidopyrine or other coal tar derivatives the drug should be interdicted. Steps should be taken to eliminate the substance remaining in the body by the use of antidotes as dimercapto propanol (BAL). If the bone marrow is seriously depressed and does not respond it is usually necessary to resort to transfusion. The hemogram should be brought back to normal and every effort made to maintain it at this level until the bone marrow recovers.

Hypochromic normocytic anemia plus history of blood loss, rapid growth or pregnancy usually in the presence of a diminished supply of iron makes the diagnosis. The administration of iron (ferrous sulfate, ferrous carbonate, ferrous gluconate, iron and ammonium citrate and reduced iron) is followed by reticular cell increase and Hb increase. This indicates that the search is to be continued for the causative lesion.

Hypochromic microcytic anemia may also occur in severe and chronic infections, sepsis, nephritis and lead poisoning but these are not of iron deficiency type.

### Pernicious Anemia

Pernicious anemia cases may be maintained quite well by feeding liver or meat digested with normal gastric juice or the stomach mucosa. All of these are effective in the dried and extract form as hepatin or Ventriculin. Highly refined products have been prepared for parenteral use. The synthetic product, folic acid and especially the new liver vitamin B<sub>12</sub>, are spectacular in restoring the blood picture to normal. Vitamin B<sub>12</sub> also halts, relieves and cures the lesions of the spinal cord and is necessary in the cases which are progressing rapidly.

Secondary treatment as the substitution for the achlorhydria and other deficiencies as hydrochloric acid and vitamin mixtures would be desirable especially if there is diarrhea. In the P.A. of pregnancy vitamin B<sub>12</sub> alone is not sufficient and folic acid must be added.

Hemolytic anemias as found in familial or acquired acholuric jaundice are found to respond to the removal of the spleen and gall bladder when it is filled with the pigment.

There are some atypical hypochromic anemias associated with tuberculosis rheumatic fever malaria and certain exotic infections, and in toxication with gold. These may rarely show only a low erythrocyte count but also usually decreased leucocytes and platelets i.e. anemia leukopenia and thrombocytopenia.

The platelets are antigenically related to the vascular endothelium. Administration of iron is essential in the treatment of iron deficiency anemia of chronic blood loss rapid growth pregnancy, G. I. defects, and hookworm disease.

Primary hypersplenism has been described in which one two or all three cell types as platelets white blood cell and red blood cell counts are reduced. This may occur secondarily after there has been a thrombosis of the splenic vein. Splenectomy may be a curative procedure in these conditions.

In Lant's syndrome the liver damage usually is considerable in that there has generally been hemorrhage from varices in the esophagus or stomach and the less heroic procedure of tying off of the splenic arteries must be undertaken.

### Lymphoblastomas

Hodgkin's leukemia lymphatic and myeloid have been temporarily favorably affected by the use of nitrogen mustard methyl bis ( $\beta$  chloro ethyl) amine hydrochloride known as  $\text{HN}_2$ . Doses of 0.1 mg per kilogram of body weight are injected daily for from 4 to 6 days. Toxic reactions as fever and aching are very commonly suffered. Radiation however still remains the most reliable form of therapy and nitrogen mustards are generally used only after the patient has become very resistant. Hodgkin's disease is a tumor rather than a granuloma.

ACTH in 25 mg doses q 6 h has been found to cause a remission in acute leukemias in only one half the cases but all relapsed. Chronic lymphomas leukemia lymphosarcomas and Hodgkin's disease were found to show regression of all lesions but no improvement in the basic pathology. A relapse was prompt after suspension of the ACTH. Remission of multiple myeloma has appeared to persist longer. Early aplastic anemia resulting from chemical intoxication has responded to ACTH in 25 or 125 mg q 6 h doses and the good effects have persisted.

tein diet rich in vitamins, methionine, and choline, and high in iron. Ligation of the splenic artery with resulting infarction of the spleen is often beneficial.

**Hemophilia** may produce anemia of acute or chronic blood loss and is probably incurable, but life may be lengthened if fraction I of gamma globulin, which has been isolated by the Boston group is available and used in the therapy. The bleeding of a hemophilic is amenable to therapy. Ecchymoses tend to develop after minor trauma, and should be prevented from extending by cutting off the circulation to the area by pressure, and by the application of cold. The injection of minute increasing amounts of histamine allows the thrombin to accumulate form fibrin, and stop the bleeding. It may be converted into a hematoma if heat is applied before the bleeding stops. Refrigeration and later manipulation should be used to prevent ankylosis of the joints. Fresh platelet potent plasma and blood should be given. Twice as much older plasma is needed.

**Polycythemia vera** has been treated generally with repeated bleeding which tends to remove the excess of erythrocytes. Induced ankylostomiasis has accomplished similar results. Depression of the erythropoietic tissue in the bone marrow and the intrinsic substance in the gastric juice by removal or by radiation has been considered promising. Radioactive phosphorus injected intravenously by experts has been shown to be the most effective remedy. It should be administered only by a hematologist with special training in the use of isotopes.

In **hemolytic anemias**, as in sickle cell anemia which is incurable, oxygen at the time of crises is good therapy. Vasodilators as niacin or papaverine may be used and a high protein intake may be of some value. Blood transfusions must be carefully given. In **familial icterus**, splenectomy is a curative in cases of familial spheromicrocytic or in some cases of acquired hemolytic jaundice. In atypical cases operation is advised only when conservative measures have failed to produce improvement. Transfusion increases the number of normal red blood cells in the circulation and may be the therapy of choice. Increased temporary resistance of the erythrocytes has been found to be improved by medical treatment with vitamins C and B, as it has also been improved by splenectomy. However, the spherocytosis persists.

The usual and unusual causes for anemia should always be considered and looked for, as nutritional deficiencies and hypersplenism as well as aleukemic leukemia or any type of leukemia, before considering the anemia as a disease entity.

**Method of Insulin Treatment**—In recent cases of diabetes and those not previously treated, give 10 units PZI the first day. Increase daily by 5 to 10 units until urine is nearly sugar free on arising or until blood sugar (fasting) approaches normal levels.

If glycosuria and hyperglycemia persist after meals add supplementary doses of insulin (varying with Benedict test), e.g. yellow, 5 units, orange 10 units and red 15 units.

Replace multiple injections with single dose admixture. Replace total with 2 regular to 1 PZI mixture. If daytime glycosuria and hyperglycemia persist, increase insulin content e.g. 2½ 1 3 1. If postabsorptive (fasting) glycosuria and hyperglycemia persist increase amount of PZI in mixture e.g. 3 2. The criteria for adjustment of admixture are identical with those governing separate doses. Mixtures are always prepared from preparations having the same number of units per cubic centimeter.

In patients who have been taking insulin replace total daily dosage with three fourths as much PZI and increase dose until urine is sugar free on arising.

The dose of PZI is regulated by the *fasting conditions* and the doses of insulin by the conditions during the daytime feeding hours. Glycosuria and hyperglycemia in the daytime call for more insulin whether it is being given separately or as one component of a mixture. If necessary during emergencies or while a patient is just becoming stabilized, separate supplementary doses of insulin may be administered in respect to meals governing the dose by the four period urine tests as follows:

Urine	Green	1+ = 5 units
Benedict	Yellow	2+ = 10 units
Qualitative	Orange	3+ = 15 units
Test	Brick red	4+ = 20 units

(If inadequate increase amounts)

All patients are given PZI before breakfast. The aim is to control patients with insulin injections once daily prior to their discharge from the hospital.

Hypoglycemic attacks are manifested by weakness, pallor, tremor, sweating, nervousness, diplopia and even convulsions and coma. In patients taking PZI or any long acting insulin preparation sweating and tremor may be absent but morning headaches, slight nausea or irritability or lethargy may be predominant. If there is any doubt the patient should be given sugar (orange juice) and if that cannot be tolerated by mouth it should be given intravenously 20 Gm. If taking PZI a large infusion of 5 to 10% glucose may be required. Any tendency to relapse should be noted for the next several hours.

## Management of Diabetes Mellitus

### MODIFIED METHOD OF PFCK

The patient with diabetes mellitus who has been brought out of coma or acidosis or who has never suffered any such complications must be carefully studied and adjusted on a satisfactory regime of management. A standard chart should be kept on every patient and all of the various entries should be completed, including history number, age, height, and weight. The weight should be recorded in the proper column at least once each week unless the patient is too ill to get out of bed. Each line of the chart is a daily balance sheet and it must be completed.

A diet is prescribed based on the following data

1 Ideal body weight in kg and the established rules based on requirements

2 Calories 25 per kg at rest in bed (elderly or obese)

30 per kg at moderate activity

35 per kg for young and active patients

3 Carbohydrate to supply 40 % of calories, protein to supply 15 % of calories (1 Gm per kg), fat to supply 45 % of calories. This will place carbohydrate and fat approximately in a 2 : 1 ratio by weight

4 Calculation e.g., Patient's ideal weight 132 lb = 60 kg

at 25 cal/kg = 1500 cal

Protein 1 Gm/kg = 60 Gm

Carbohydrate = 40 % of 1500 cal = 600 cal / 4 = 150 Gm

Fat = one half of carbohydrate = 150/2 = 75 Gm

The diet will be C 150 P 60 F 75 = 1515 cal, regular soft or liquids

5 The minimum routine orders for every patient with diabetes are

(1) Blood sugars fasting and 4 P.M. postprandial every Monday and Friday. See that these are drawn and sent to laboratory promptly so that the report may be properly recorded on the chart

(2) Period urine specimens to be examined every day. Fasting (before breakfast) 11 A.M., 4 P.M., 10 P.M. Record test daily

Special orders will vary with each case. Blood sugar estimations and other examinations such as acetone diacetic acid CO<sub>2</sub> NPN should be made without delay when indicated

Regular insulin is indicated in all diabetic patients with complications such as infections, surgery, pregnancy, coma and who have proved their inability to maintain normal weight and strength without hyperglycemia and glycosuria. PZI is basic for all cases. Mixtures of insulin and PZI are used when total doses are more than 30 units daily and in multiple doses cases

**Method of Insulin Treatment**—In recent cases of diabetes and those not previously treated, give 10 units PZI the first day. Increase daily by 5 to 10 units until urine is nearly sugar free on arising or until blood sugar (fasting) approaches normal levels.

If glycosuria and hyperglycemia persist after meals add supplementary doses of insulin (varying with Benedict test) e.g., yellow, 5 units orange, 10 units and red 15 units.

Replace multiple injections with single dose admixture. Replace total with 2 regular to 1 PZI mixture. If daytime glycosuria and hyperglycemia persist increase insulin content e.g. 2½, 1, 3 1. If postabsorptive (fasting) glycosuria and hyperglycemia persist increase amount of PZI in mixture e.g., 3 2. The criteria for adjustment of admixture are identical with those governing separate doses. Mixtures are always prepared from preparations having the same number of units per cubic centimeter.

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All patients are given PZI before breakfast. The aim is to control patients with insulin injections once daily prior to their discharge from the hospital.

Hypoglycemic attacks are manifested by weakness pallor tremor sweating nervousness diplopia and even *convulsions and coma*. In patients taking PZI or any long acting insulin preparation sweating and tremor may be absent but morning headaches slight nausea or irritability or lethargy may be predominant. If there is any doubt the patient should be given sugar (orange juice) and if that cannot be tolerated by mouth it should be given intravenously 20 Gm. If taking IZI a large infusion of 5 to 10% glucose may be required. Any tendency to relapse should be noted for the next several hours.



All patients should be instructed in the rudiments of diabetes control. Joslin Manuals are available for patients in the wards. As soon as the patient is physically and mentally able, the dietitian should begin the dietary instruction. He should be taught how to examine urine with Benedict's solution and he should be supervised until he is proficient. He should know how to record his tests, how to save a 24 hour urine sample and a 4 ounce specimen. Under the nurse's supervision, each patient should learn how to sterilize his syringe, measure his dose of insulin and PZI, make the proper admixture, and administer his own dose.

Reference Peck, E. P. Indianapolis General Hospital. Eli Lilly Laboratory of Clinical Investigation. Personal communication.

### The Use of Insulins in Diabetes —

Protamine insulin is indicated for use in over one half the cases of diabetes. Its advantages are that it need be injected only once daily, is painless on injection, and maintains a gentle insulin effect without violent fluctuations in glycemia. The patients in whom protamine insulin is indicated are mild diabetics, older diabetics, obese people, stable forms of diabetes, when there are no allergic tendencies, those in whom dosages under 40 units of insulin daily are given, when the fasting sugar levels are high in proportion to the postcibal glycosuria, and when globin insulin tends to cause afternoon insulin reactions.

Globin insulin or protamine insulin mixtures are indicated in severe diabetics, young diabetics, thin people, where insulin dosage is more than 40 units daily, where postcibal glycosuria is heavy in relation to fasting blood sugar levels, in labile forms of diabetes when protamine insulin tends to cause nocturnal insulin reactions, or where allergic manifestations exist. Globin insulin often causes stinging on injection which is rather a disadvantage.

**NPH 50** neutral crystalline protamine zinc insulin (Eli Lilly & Company). This new product of pH 7.2 as contrasted to the acid pH 3.0 of regular insulin has been developed by Hagedorn and his associates who crystallized protamine zinc insulin. N refers to neutral, P to protamine, H to Hagedorn and 50 to 50 mg. of protamine used in the preparation of 100 units. The action is intermediate between regular and protamine zinc insulin, continuing for at least 24 and probably for 28 or 30 hours. The maximum effect develops during the late afternoon, evening and night. To compensate for this, some of the carbohydrates from breakfast are shifted from breakfast to afternoon and bedtime lunches. A single daily before breakfast dose of NPH 50 should replace separate injections of regular and globin or protamine zinc insulin.

## Maintenance Therapy in Addison's Disease

(Peter Bent Brigham Hospital)

The patient with Addison's disease who has been brought out of a crisis or who has not suffered any such complication must be given a fixed daily supplement of sodium chloride (enteric coated tablets). Four grams of NaCl are given daily while desoxycorticosterone acetate requirement is being determined. If edema or hypertension occur after implantation the supplement of NaCl should be decreased. During hot weather or in the presence of infection or if signs of pellet depletion occur it should be increased.

The minimum daily requirement by subcutaneous injection of desoxycorticosterone acetate (parenterally) to maintain weight, blood pressure and well being is 0.5 to 1.5 mg. determined during a four to six months period of observation.

The pellet requirements are one 125 mg. pellet implanted for each 0.5 mg. required by injection. The implantation of pellets is carried out in the operating room. Each pellet is implanted in a separate subcutaneous pocket in the subscapular region. A small amount of cortical extract is given on the day of implantation and for a few days after. The pellets usually last twelve months.

Desoxycorticosterone acetate is a very potent hormone and there are dangers inherent in its action. All patients who receive this substance should be carefully observed for increase in blood pressure, retention of salt and water and increase in size of heart.

Reference: Thorn G. W. Personal communication

## Management in Patients With Heart Disease

Patients with organic cardiovascular diseases may usually be maintained in circulatory equilibrium by careful management. Evident etiological factors if still active as hypertension, infectious disease, hyperthyroidism, avitaminosis or arteriovenous aneurysm should be treated and removed if possible.

Known perpetuating factors as obesity, diabetes, goiter, overeating and overwork should be therapeutically attacked, and precipitating factors as sudden rains, exertion, emotion, excitement, exposure, overeating and infections should be avoided.

The patient who has impending congestive failure or in whom edema has developed and has been relieved must be maintained on a strict regime consisting of a low salt diet with adequate fluid intake 2500 to 3000 cc. The low salt diets are described in Part V under Dietetic Methods. Usually in addition to this the patient needs cardiac restorative therapy.

in the form of *coronary vasodilators* as niacin 50 to 100 mg, niacinamide 100 to 200 mg, khellin 40 mg t i d, or aminophylline 0.5 Gm in suppositories twice daily and supportive therapy as *Digitalis* P. L. 0.1 Gm daily. As a rule these patients require *diuretics* at weekly or biweekly intervals with, at times augmenting acid salts.

In addition to being salt free, the *diet* should be high in vitamins and supplementary vitamin should be given. Natural *laxatives* as Mucilose from *Plantago ovato* will help the patient keep comfortable, minimizing distention and constipation. The cardiac patient must be taught not to strain at stool. In the acute attacks, morphine, papaverine, and ouabain are needed as well as bloodless phlebotomy, venous section may be employed as described in emergency measures in the treatment of heart failure. *Rest* in the most comfortable position is to be secured for all cardiacs and when compensated, at least 14 hours of each day is to be spent in the reclining position. *Sedatives* are often desirable, particularly in hypertension cases.

Digitalization is routinely carried out in all patients who have had myocardial insufficiency and may be carried out successfully in any number of ways by Cedilanid 16 mg, Digoxin, 15 mg or digitoxin 12 mg intravenously or 16 mg by mouth, in one dose or divided dose. The mass of these is so small that it does not cause irritation to the stomach. Lanatoside C alone or in combination with digilanids A, B and C may be used in a similar way intravenously or by mouth with less danger of toxic reaction. Digitoxin or digilanid may be given in 0.2 mg doses every 6 hours for 4 to 6 doses the first day and for a second day then continued in the maintenance dosage. Digitoxin 0.1 or 0.2 mg and digilanid 0.2 or 0.4 mg daily are the maintenance dosage. *Digitalis* leaf powdered and put in pills may be given as 0.1 Gm doses and 0.2 Gm doses t i d for several days and then maintained at 0.1 Gm doses daily thereafter. A maintenance dose of *digitalis* leaf is usually substituted after digitalization has been accomplished.

*Diuresis* is usually necessary to relieve gradually accumulating salt and fluid retention in pulmonary and other dependent tissues. It is usually desirable to give acid salts for a 24 hour period before the diuretic is administered. Ammonium chloride in 1 Gm doses every 4 hours for 4 to 6 doses (4 to 6 Gm) (60 to 90 gr) is given by mouth. If the renal function is not significantly impaired an organic mercurial diuretic is chosen. Thiomerin 40 mg Hg per c c is the safest and most easily administered as it is well tolerated subcutaneously in 1 to 4 c c doses usually 2 c c are given. Mercurhydrin is tolerated intramuscularly but Mercuzanthin and Salyrgan must be given intravenously and are there

more hazardous. The mercurial diuretics are usually tolerated quite well as often as every third day but should be spaced as much as possible and given whenever the weight increases.

### Methods of Treatment of Peripheral Vascular Disease

The indications in patients with peripheral vascular diseases are for arterial dilatation and the opening up of collateral arterioles. The management follows three different lines: physical, medical and surgical, all of which are often combined. Heat must be used with care as it increases local metabolism. Refrigeration with ice has been used to conserve tissues. Alternating and contrasting hot and cold baths sometimes stimulate circulation. Diathermy and microwave therapy, iontophoresis with Mecholyl (acetyl beta methylcholine) have been used.

Massage, postural exercises and alternating suction and pressure in a glass boot, *pavlex* or passive vascular exercise therapy with negative environmental pressure atmosphere of 80 to 100 and positive pressure of 10 to 20 mm. Hg developed by Dr. Louis G. Herrmann of Cincinnati have been most successful. The indications for *pavlex* therapy are: Acute arterial occlusion due to embolism, thromboses, traumatic lesion or surgical ligation; acute peripheral circulatory stasis from frost bite or chilblains; arteriosclerosis obliterans in major arteries and secondary arteries; early thromboangitis obliterans with circulatory insufficiency due largely to secondary vasospasm. The benefits from *pavlex* will be in direct proportion to the number and distensibility of the arterioles and smaller arteries which are unaffected by the disease process.

The contraindications to *pavlex* therapy are: active thrombophlebitis, cellulitis, acute or subacute arteriolar obliteration, capillary thrombosis, advanced thromboangitis obliterans or advanced arteriolar sclerosis with capillary stasis.

The *vasoscillator bed* provides a passive mechanical device for using blood mass and gravity for vasodilatation.

Medical vasodilatation is accomplished by alcohol, xanthine derivatives as theobromine and sodium salicylates or aminophylline, iodides, nitrates and papaverine are also used but best results have been accomplished with nicotinamide, Priscoline and khellin.

Fever production by vaccines, proteins or soluble bacterial pyrogen (Pyromen, Don Baxter Inc.) or by blankets with insulation or immersion of a part in hot water  $44^{\circ}$  to  $47^{\circ}$  C will relieve vasospasm.

Anesthetic block, Novocain or alcohol injection, surgical sympathetic ganglionectomy, venectomy and arteriotomy are resorted to or considered before resorting to amputation.

in the form of *coronary vasodilators* as niacin 50 to 100 mg, niacinamide 100 to 200 mg, khellin 40 mg t i d, or aminophylline 0.5 Gm in suppositories twice daily and supportive therapy as *Digitalis* P L 0.1 Gm daily. As a rule these patients require *diuretics* at weekly or bi weekly intervals with, at times, augmenting acid salts.

In addition to being salt free, the *diet* should be high in vitamins and supplementary vitamin should be given. Natural *laxatives* as Mucilose from *Plantago ovato* will help the patient keep comfortable minimizing distention and constipation. The cardiac patient must be taught not to strain at stool. In the acute attacks morphine, papaverine, and ouabain are needed as well as bloodless phlebotomy, venous section may be employed as described in emergency measures in the treatment of heart failure. *Rest* in the most comfortable position is to be secured for all cardiacs and when compensated at least 14 hours of each day is to be spent in the reclining position. *Sedatives* are often desirable, particularly in hypertension cases.

Digitalization is routinely carried out in all patients who have had myocardial insufficiency and may be carried out successfully in any number of ways by Cedilanid 16 mg, Digoxin 15 mg or digitoxin, 12 mg intravenously or 16 mg by mouth, in one dose or divided dose. The mass of these is so small that it does not cause irritation to the stomach. Lanatoside C alone or in combination with digitanids A, B, and C may be used in a similar way, intravenously or by mouth with less danger of toxic reaction. Digitoxin or digitanid may be given in 0.2 mg doses every 6 hours for 4 to 6 doses the first day and for a second day, then continued in the maintenance dosage. Digitoxin 0.1 or 0.2 mg and digitanid 0.2 or 0.4 mg daily are the maintenance dosage. *Digitalis* leaf powdered and put in pills may be given as 0.1 Gm doses and 0.2 Gm doses t i d for several days and then maintained at 0.1 Gm doses daily thereafter. A maintenance dose of *digitalis* leaf is usually substituted after digitalization has been accomplished.

*Diuresis* is usually necessary to relieve gradually accumulating salt and fluid retention in pulmonary and other dependent tissues. It is usually desirable to give acid salts for a 24 hour period before the diuretic is administered. Ammonium chloride in 1 Gm doses every 4 hours for 4 to 6 doses (4 to 6 Gm) (60 to 90 gr) is given by mouth. If the renal function is not significantly impaired an organic mercurial diuretic is chosen. Thiomerin 40 mg Hg per c c is the safest and most easily administered as it is well tolerated subcutaneously in 1 to 4 c c doses usually 2 c c are given. Mercuhydrin is tolerated intramuscularly but Mercuzanthin and Silyrgan must be given intravenously and are there

The feet should always be kept warm. Loose fitting bed socks in stead of mechanical heating devices or hot water bottles are preferable. In the mornings the feet should be wiped free of any remaining oil from the preceding evening and dusted with plain talcum or Vioform powder over and between the toes. Use a clean pair of socks each day. Woolen socks may be worn in winter and white cotton socks in warm weather. Do not wear circular garters and do not sit with crossed legs. Shoes should be carefully fitted with special care that they are not tight. They should be made of soft leather and without box toes in order to prevent pressure and trauma.

The toenails should be cut straight across and only after the feet have been thoroughly cleansed. The light should be good. Corns and callouses should not be cut.

Reference: Herrmann, L. G. *Passive Vascular Exercise* Philadelphia 1936 J. B. Lippincott Co. and Personal communications.

### Principles in the Dietary Management of Peptic Ulcer

Acute ulcer in the stomach or duodenum may erode a blood vessel and the bleeding stage creates an emergency state. Confinement to bed and sedation with Demerol to suppress gastric motility and atropine to reduce spasm and gastric secretion are followed by institution of frequent small feedings of the Meulengracht, the Andresen or the proteolytic diets which are described in the pages that follow. If the blood loss is considerable or significant it should be replaced by whole blood transfusion especially if the blood pressure drops below critical levels and the pulse rate rises unduly. Parenteral feedings are at times resorted to but the gastric motility must then be quieted by drugs.

Medical treatment in general must be directed toward neutralizing and suppressing the excessive secretion of acid gastric juice by food alkalis and atropine. The increased gastric muscle tone or spasm and contractions of hunger must be relieved by very frequent at least hourly feedings and by antispasmodics.

Foods should be liquid, soft, tasty, well cooked and finely divided as strained or pureed. The basic foods in ulcer diets have been dairy products but for those who tolerate fats poorly or who are obese butter, cream and egg yolks are omitted. Milk however is still the chief food substance in most ulcer diets.

Low fat diets are being more and more considered replacing animal fat with soybean whipped custard, gelatin, white softened bread and toast, skimmed milk soups, pureed vegetables, mashed or mealy baked potatoes, scraped or finely ground lean meats or dry fishes and as desserts applesauce or fruit pastes, jellies and Jello.

### Urgent Therapy in Peripheral Arteriolar Disease

Intravenous therapy is resorted to in severe cases. Two courses of 10 to 12 days each are given with rest periods of from 2 to 4 days between. If relief is not obtained, a third course is instituted. Skin temperature and oscillographic readings are taken at the end of each course. The solutions used are as follows:

Buffered 2 % sodium citrate solution or

Ether (anesthesia quality) 2.5 % in 1000 cc of M/6 Na lactate or 5 % glucose, or 0.9 % sodium chloride or

Intra-arterial histamine acid phosphate 0.275 mg in 250 to 500 cc normal saline or 5 % glucose or

Alcohol 5 % in 5 % glucose

### General Measures

Tobacco in all forms is absolutely interdicted.

**Diet**—The dietary should be high in vitamins and calcium. It should contain plenty of green vegetables and fruits in an otherwise well balanced liberal diet, unless some special diet has been prescribed. The fluid intake should be kept high at least four quarts of water each day.

**Exercise**—*Directions* Recline with shoes and stockings off. Raise both legs vertically into the air bending them at the hips so that the soles of the feet face ceiling. If this position is painful or impossible the legs may be raised as nearly vertical as possible or rested against a wall to the side of the bed. Keep legs raised exactly one minute by the clock. Lower the legs and sit up in bed with the legs hanging freely over the edge of the bed not touching the floor. Keep the legs in this position for exactly one minute by the clock. While they are hanging over the edge of the bed the feet should be moved slowly back and forth and from side to side at the ankle joint. Now lie down flat in bed rest for one minute in this position. The process is to be followed through six times in all.

**Care of the Feet**—The feet should be washed each night with neutral (face) soap and warm water and dried with a clean soft rag without rubbing the skin. Apply 70 % rubbing alcohol and allow the feet to dry. Gently massage the skin of the feet with petroleum jelly, cocoa butter or lanolin, generously applied. Epidermophytosis and infection between the toes should be controlled with Vioform powder during the day and ointment at night. Do not use strong antiseptic drugs such as tincture of iodine, Lysol, cresol or carbolic acid on the feet. At the first signs of a blister, infection of the toes, ingrowing toenail or trouble with bunions, corns, or callouses, a doctor should be consulted.

### Management of Liver Disease

Liver cell damage and consequently functional disorders result from such etiological factors as biliary obstruction severe infectious diseases and metabolic disorders as thyrotoxicosis and specific hepatotoxic substances such as chloroform and carbon tetrachloride. These agents produce similar pathological changes of varying degree with progression and regression at varying rates to healing and repair. Nutritional disorders incident to secondary starvation from protein, especially choline deficiency diet anemia diabetes mellitus and subacute infection result in heavy fatty infiltration and degeneration is the dominant pathological picture.

Fatty infiltration may persist and contribute to liver cell atrophy and fibrosis result. Simple atrophy condensation of the cords and necrosis and destruction extends out from the central vein often to the periphery with subsequent repair. Fibrosis and regeneration involving the region of the central vein and progressing toward the periphery of the lobule make up the pathological unity of liver disease.

Repair and regeneration generally are not inaugurated in the presence of (1) alcoholism and protein choline or vitamin B deficiency (2) complete obstruction of the bile duct (3) the persistence of the hepatotoxic agent or (4) when there is a marked reduction of the flow of blood. Recognition and correction of these factors is in order.

**Cirrhosis of the Liver**—It is of course desirable from the therapy point of view to suspect and diagnose cirrhosis in its incipient and fatty stages when only a few gastro intestinal symptoms and little if any ascites are present and when the liver pathology is still easily reversible. Steps should be taken to minimize the work of the liver and to decrease or eliminate any source of intoxication. In the dietary condiments are of course taboo as are also smoked meats smoked fish tough meat coarse vegetables and cereals. A strict lactovegetarian regime may be too enervating and monotonous but skimmed milk buttermilk and acidophilous milk and cottage cheese should be taken freely. Alcohol substitutes calories and leads to protein especially choline deficiency.

A high protein high carbohydrate and low fat diet with an excess of vitamin B has been advocated with the oral and intravenous use of methionine choline brewer's yeast and glucose. Insulin is in order to increase the glycogen content of the liver. Forty to fifty grams of glucose in the form of Karo corn syrup is given followed by 20 to 30 units of insulin given subcutaneously. Restrictions in the diet should be minimal because of the difficulty of keeping the nutrition up in these patients. A diet low in water and sodium content as an anti-ascetic



Peptic ulcer diets are given in detail in Part V, Dietetic Methods

Antacids and antispasmodic drugs and sedatives are important adjuncts. Rest, relaxation, regulation of habits, protection from tension states, emotional stress and strains, and psychotherapy are necessary not only for the period of acute symptoms but for all time. The patient must learn to live with his ulcer and with himself and those about him, and relax mentally as well as physically.

Constipation must be overcome by natural laxatives as fruit pulps or hydrophyllic sterculin gums of *Plantago ovata*, Metamucil, Mucilose, etc., rather than mineral oil and agar emulsion. Every stool should be described.

The use of tobacco in any form, alcoholic beverages, and condiments must be absolutely prohibited. A patient who continues to smoke or drink cannot heal his peptic ulcer. The symptoms should be relieved, relapses postponed, and complications prevented.

An intractable ulcer is usually a complicated ulcer, therefore, commonly associated disorders, if present, should be treated. Spastic colon, subacute appendicitis, or cholecystitis, recurrent infections, tonsillitis, pharyngitis, sinusitis, and glandular dysfunction as hyperthyroidism should receive medical attention.

Family troubles must be ironed out, financial worries removed in so far as is possible. Environmental difficulties, emotional conflicts, psychic problems, and chronic tension states must be relieved.

The physician must become the confessor and advisor as well as disciplinarian, teacher and dietitian who will prescribe bland diets low in residue, given in small feedings at regular and frequent intervals, a vacation if possible with rest away from the usual haunts, or sedatives as Solofon or Delval, or phenobarbital regularly to control and prevent the jitters, alkalies as magnesium oxide or trisilicate, aluminum hydroxide as Amphojel or Phosphaljel Gelusil, or combination, anti-spasmodics as belladonna, atropine, Novatrine, Syntropan, Pavatrine, or Trasentine, with hot abdominal stupes, gastric aspiration and lavage.

Banthine (Searle), a new anticholinergic drug has been shown by Grimson and collaborators to be very effective in facilitating healing of peptic ulcer. Doses of 100 mg q 6 h day and night for 3 to 6 weeks.

Repeated hemorrhage, perforation and high grade obstruction and malignant degeneration are complications to be guarded against and when they develop, surgical intervention must be conceded.

Reference Grimson K S, Lyons C K, and Reeves R J. J A M A 143 873  
July 8 1950

### Management of Liver Disease

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measure has not been successful in our hands, nor has the routine injection of mercurial diuretics given such spectacular results as have been reported. In fact, the only patients in whom we have had great success with mercurials are those in whom syphilis was the etiological factor along with alcoholism in the production of hepatic cirrhosis or in whom heart failure, with sodium retention, had contributed to the ascites.

Magnesium sulfate, with or without peptone in glycerinated water, may facilitate the expulsion of bile from the biliary tract or from the gall bladder, and at the same time alleviate the constipation. Sometimes mineral oil and agar or Mucilose are also necessary. Animal charcoal and bolus alba have been given more or less empirically to remove toxic substances from the intestines. Silver chloride and silicic acid have been recommended for accomplishing the same absorption but are not widely used.

All of those substances which expel bile from the bile passages may be given with impunity. The choleretic substances, particularly dehydrocholic acid (Decholin) preparations, must be given with great care if there is obstructive jaundice. In mildly jaundiced individuals choleretic substances, biliary acids especially Decholin, and less effective saheylates and oil of peppermint, may favor the secretion of bile. Other chologues are of doubtful value. The absence of free acid and pepsin from the gastric juice calls for the administration of the digestive substances HCl and pepsin after each meal.

Biliary cirrhosis caused by the infection or obstruction of the biliary passages a common accompaniment of cholecystolithiasis, may be temporarily benefited by duodenal drainage with magnesium sulfate, but, in general, surgical drainage methods are necessary. Biliary cirrhosis, of course with external obstruction to the outflow of the bile may be remedied by the release of the obstruction when this is possible. Surgical treatment should not be undertaken in individuals in whom the liver has been so damaged that as much as 30% of the Bromsulphalein has been retained in the blood stream after 30 minutes.

The development of ascites may be controlled particularly in a syphilitic patient by the use of mercurial diuretics but sooner or later one must resort to tapping under surgical or aseptic conditions. A peritoneal subcutaneous button has been developed to relieve ascites. Surgical treatments directed toward the increasing of collateral circulation of the liver or the removal of the spleen have been doubtful certainly not spectacular, successes. However ligation of the splenic artery has resulted in clearing of the ascites and considerable improvement in many cases. In the Talma Morison operation, the mortality is as high

as 30 % and even in the best clinics it has never been below 15 % The implantation of the omentum in the groin and around the femoral subcutaneous veins has been done successfully The Mayo modification of bringing the omentum up behind the rectus muscle in the posterior sheath of the abdominal wall has done little good

Venous anastomotic shunts between the portal veins and renal or splenic left renal vein anastomoses have been reported successful in some cases The flow of blood retrogradely through the superior gastric or coronary veins to esophageal plexus should be interrupted by ligating as completely as possible in individuals who have had hemorrhage from esophageal varices Such a procedure may be lifesaving The injection of the esophageal varices with sclerosing solution by working with a long needle and through an esophagoscope has been successful

In Banti's disease splenectomy has been considered to be of value particularly if it is done before the stage of ascites in other words before the portal cirrhosis has developed In some cases of biliary cirrhosis improvement has followed splenectomy An occasional case of portal cirrhosis is benefited by splenectomy but the great majority of cases are entirely subjected only to ligation of the splenic artery have shown most encouraging results Operation should be considered when the ascites no longer responds to mercurial diuretics combined with adequate treatment

It is of course possible that there may come a time when preventive treatment and prevention will be considered but such is certainly not the case now for most of the cases of cirrhosis are diagnosed only after ascites develops If there must be an hereditary predisposition to liver cirrhosis it may well be that this predisposition is not primarily liver inferiority so much as it is a cortical inferiority

The histopathology of the liver may be demonstrated in tissues removed by liver biopsy

### Derangement in Functions of the Liver

The status of the pathological physiology may be revealed by the various liver function tests The prophylactic protection of the liver parenchyma has been accomplished experimentally with carbohydrate foods and proteins rich in choline and methionine Purine and carbohydrate metabolism are disturbed somewhat and a hypoglycemia may result and the levulose and the galactose tolerance tests give evidence of carbohydrate disturbance in hepatic damage

The defective deamination of the amino acids by the liver may be detected by the presence of a high blood NPN and uric acid concentration in the blood plasma with a relatively low urea nitrogen

Hypercholesterolemia may be present in the blood stream when the liver is fatty. Frequently, however, there is found a decrease or even a very sharp lowering of the blood fats and cholesterol esters as lecithin.

Bilirubin excretion is interfered with and may be demonstrated by injecting bilirubin, the substance itself, directly into a vein and timing its persistence in the blood stream. Likewise tetrahalogens as phenol, sulphonephthalein, Iododekin or Bromsulphalein may be retained in the blood stream for an abnormally long time after injection in the presence of liver damage.

The liver is a great storehouse for vitamins A, B, B<sub>1</sub>, B<sub>2</sub>, C, D, complex, and for vitamin K particularly. The obstruction of the bile flow into the gut adversely affects the absorption of the vitamins.

The conjugation and possibly detoxifying power of the liver is demonstrated in the change of benzoic acid to hippuric acid. This has been used as a test of liver function.

The blood proteins are in part at least built up in the liver. The serum albumin is found to fall as a rule in the presence of liver damage and there is a compensatory attempt to produce a globulin elevation which is often considerable but usually insufficient. The albumin globulin ratio is reversed. The hypo or oligoproteinemia decreases the plasma osmotic pressure as much as 50 %.

The typical increased portal and decreased plasma osmotic pressure are not, however, alone at work in the production of the ascites of portal cirrhosis. Injury of the capillary walls in the serosa and the increased permeability of the tissues in general, as well as the capillaries, allow transudation.

Some of the metabolic disturbances show at the time of liver enlargement before the fibrosis and contraction have occurred. Hepatic failure, however, may come on during these early stages and result in 'cholemia' which is the result apparently of failure of the detoxifying mechanism of the liver.

In hepatic disease there is an anoxemia and a lowering in the sodium chloride level of plasma, an acidosis, and an increase in lactic acid and citric acid. It is suggested that the liver is the storage reservoir for chloride, and the possibility of its having a hormonal influence on the chloride has been considered but not proved.

Anemia results when disease of the liver causes decreased affinity of hemoglobin for oxygen and displaced the oxygen disassociation curve to the right. Whether this is due to the change of hemoglobin or the mixture of interfering substances is not known. There is a concentration of the blood and a macrocytic anemia in hepatic cirrhosis.

A macrocytic anemia of a most obstinate type develops due to the failure of the liver to store the active hemoglobin building principle or its inability to present it in the form for utilization by the body. This would suggest the necessity for huge doses of iron as well as liver extract with transfusion.

Hemorrhage is the result of (1) rupture of the collateral circulatory channels or (2) the specific change in the prothrombin content of the blood. Profuse bleeding from the esophageal varices is a surgical problem. Ligation of the splenic artery or splenectomy in Banti's disease removes a goodly volume of blood from the portal vein and is usually beneficial. Furthermore ligation of the coronary veins of the stomach has been attempted usually in vein and hemorrhage cases of cirrhosis of the liver. However ligation of the splenic artery has been successful in removing much of the portal volume and stimulating splenic adhesions.

Cholemic bleeding is most likely the result of a low prothrombin content in the blood and proved by Hawkins and Brunhouse to be the case in experimental animals with biliary fistula and the hemorrhagic state cured by feeding bile salts. Then too Dam and his co-workers showed that the hemorrhagic state in chicks receiving adequate vitamins A, D, D<sub>2</sub>, and C but low K were cured by an addition of the coagulation or K vitamin found in alfalfa or from the unsaponifiable nonsterol fraction of a liver fat. Dicumarol was found to produce in animals a hemorrhagic condition such as follows the taking of spoiled sweet clover which Roderick and Quick has shown to be dependent upon low prothrombin level and pathologically these animals have had focal necrosis of the liver. Therefore one must consider that the prothrombin is reduced in hepatic insufficiencies of the biliary obstructing as well as liver damage of other etiology. The bile salts make possible the absorption from food of the natural K vitamin and stop the hemorrhagic tendency.

Treatment is not a simple matter it must be administered with the complete pathological physiology of hepatic insufficiency in mind. First, alcoholism any factor of malnutrition and any obstruction in the biliary tract area must be obviated. Second any hepatotoxic agent must be removed. Third the blood flow to the liver must be improved with changes in the general as well as with portal circulation.

Transfusion of 200 cc of whole blood relieves the anoxemia and should be used. High carbohydrate high protein high yeast and no fat diet containing 250 Gm with a high percentage of glucose xanthines adenine and choline is desirable to protect the liver from further damage. Choline methionine or inositol administration seem rational substitution therapy where there is muscle wasting. Choline gluconate chloride

carbonate, or citrate 6 to 12 Gm, or methionine 3 to 6 Gm, and inositol 3 to 6 Gm per day, with or without the high protein diet, will cause the fatty infiltration to clear up

Proteins of good biological character, such as delicate meats like chicken, fish dairy products, eggs protolysates of various types and Brewers' yeast, should be given in amounts of 1 up to  $1\frac{1}{2}$  Gm per kilo gram of body weight per day. This is necessary in order to build up the serum protein. Infusion of blood solutions fortified with concentrated blood proteins may accomplish this

Fats should be given very sparingly. All vitamins should be forced orally and parenterally. Brewers' yeast 2 ounces orange juice 12 ounces crystalline vitamin B<sub>1</sub> 4 to 10 mg soluble vitamin B complex vitamin P, and crude concentrated liver extract intravenously daily may be helpful in the metabolic restoration of the liver. (See section on Diet)

#### References

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Patek, A J, Jr, and Post J. J Clin Investigation 20 481 501 Sept, 1941

### Treatment of an Irritable Colon

Diet should be adapted to the requirements of the individual patient. As a rule, for those producing a great deal of mucous and constipated stools, a bland diet high in vitamins containing a minimum amount of roughage or cellulose should be advised. Hydrophilic gums may be tried

Purged vegetables soups, mashed potatoes rice, cottage cheese custard Jello and sherbet usually suffice and give a variety with feedings at intervals of two hours. Milk may be given if well tolerated

After at least two weeks on the bland diet small amounts of cooked vegetables, carrots asparagus tips mashed potatoes and cooked fruits such as baked apples canned peaches and pears, and butter may be added

After the third and fourth weeks the pulp of uncooked foods, fruits and the less woody vegetables may be taken, meat need not be added as long as the diet is well balanced, but add chicken fish, eggs cottage cheese cooked cereal junket, malt cod liver oil and yeast and small amounts of sugar. Pork, sausage shell fish condiments and alcohol should be abstained from

Belladonna, atropine, or homatropine brommethyle (methylatropine, or Novatropine) are indicated to relieve the spasm. Belladonna should be started in 5 minim doses three times a day and gradually increased to about 30 minims three times a day until the desired physiological action is obtained at which time it may be temporarily discontinued and later resumed. Atropine may be given in 1/150 to 1/100 gr

doses three times a day while Novatropine is only one tenth as potent and one thirtieth as toxic, therefore it may be used in 1/20 to 1/10 gr two or three times a day Bromides and barbiturates may be added for exceedingly nervous individuals

Such regime of therapy will suffice in practically all cases mixed with good doses of psychotherapy and reassurance

In rare instances some advocate the method of Soper to overcome persistent spasms of the colon in the region of the sigmoid This should be tried only after the other method has been given a thorough trial The procedure is as follows

The proctoscope is slowly and carefully passed may relieve the spasm and is allowed to remain in situ for 10 or 15 minutes to admit the dilatation of the spasmodic muscle of the sigmoid A colon tube is then passed through the funnel and the scope the scope withdrawn and 60 cc of 30 % solution of magnesium sulfate forced through the tube into the colon by means of the funnel The patient retains a knee chest position for an additional 10 minutes and 200 cc of equal parts of cottonseed oil and warm castor oil are then inserted into the colon and the patient instructed to retain this as long as possible Magnesium sulfate produces relaxation of the sphincter the cottonseed oil is for lubrication the castor oil combines with sodium salt producing sodium ricinoleate and an antiseptic and play the part in combatting the secondary infection in the colon which is a fairly common complication

This treatment may be repeated every three or four days for two weeks and then given once a week for two months meanwhile the patient is allowed an occasional salt water enema or olive oil enema twice a week Plain mineral oil at night is beneficial Charcoal may be given for the gaseous distention Castor oil every ten days is helpful if the patient has loose stools containing much mucus Calcium gluconate bismuth subcarbonate and bolus alba are sometimes used when the stools are numerous (See Part V Dietetic Methods for bland diets)

### Management of Renal Stones

According to the recent work of Short an important advance in the control of renal stone is the use of Amphojel which reduces urinary excretion of phosphate and estrogens which decrease the calcium content of the urine and increase the citric acid content Together these effects help prevent or dissolve the following kinds of stones calcium phosphate calcium carbonate magnesium phosphate magnesium ammonium phosphate and calcium magnesium ammonium phosphate

Amphojel is given in 30 to 40 cc amounts four times per day one hour after meals and at bedtime This precipitates the phosphate in



the intestine in sufficient amounts to reduce urinary phosphorus excretion from 1 Gm to 150 mg per day or less

Estrogens in male patients are given in daily doses of 8 to 16 Conestron tablets which increase urinary citrate excretion 250 to 300 mg per day. After four weeks, medication should be temporarily withdrawn so that the undesired effects as reduction of libido and sensitivity of the nipples may subside. In female menopausal patients, 8 to 10 Conestron tablets are given daily for three weeks and then the drug should be withheld for ten days to allow for regression of the endometrium. Only severe withdrawal bleeding is a contraindication. In menstruating females, 4 to 6 Conestron tablets are given from the fifteenth through the twenty fifth day of the cycle.

Diet consists in excluding foods high in phosphorus such as nuts, dried apricots, barley, rice, whole grain cereals and breads, hard cheese, cocoa, sweetbreads, kidneys, heart, liver, fish roe, sardines, smelt, bovril, gelatin, and yeast. Limit such foods as milk, eggs, meat, and fish.

Reference Shorr, E. J. Urol 53: 507-520, April, 1945

### *Dietetic Management of Nephritis*

Principles governing the diet in nephritis are based upon need for rest of the damaged organ. The work required of the kidney should be reduced to a minimum by limiting the intake of substances, particularly the proteins and salts, which contribute to the mass of waste products the excretion of which is the chief function of the kidneys. The kidneys of a patient on a diet of 120 Gm of protein will excrete a total of 70 Gm of solids in 24 hours. These solids are made up by urea 32 Gm, hippuric acid 10 Gm, chlorides 14 Gm, nitrogen and other substances 3.2 to 5.5 Gm, and other salts phosphates, sulfates, oxides of potassium, magnesium, and calcium 7.5 Gm.

The average urinary output of nitrogen during starvation amounts to 7.0 to 10.0 Gm in 24 hours. The ordinary nitrogen excretion of a normal patient on a moderate protein diet of 100 Gm is 16 Gm in 24 hours. Starvation reduces the work of the kidneys but high carbohydrate feeding may depress the urinary nitrogen still further according to the principle of the protein sparing property of carbohydrate. This may decrease the nitrogen output 50 % if the carbohydrate intake is forced. The end products are carbon dioxide and water which are excreted wholly or partially by the lungs. The skin and the lungs together normally excrete as much water as the kidneys. A damaged kidney may have difficulty in excreting salt and water. Usually an increased volume of water is necessary because of the inability of the

kidneys to concentrate the urine. The intake of sodium chloride or salt in the diet should therefore be limited and at the same time care should be taken not to produce depletion.

All proteins are alike in so far as the kidneys are concerned. All contain principally nitrogen in about the same quantity whether in the form of white meat or red meat, fish, milk, whey, cheese, dried beans or peas. Urea is formed irrespective of the type of protein and makes up 50 % of the excreted urinary nitrogen. Quantity rather than the quality of protein is most important and must be restricted; however a minimum of 40 Gm. of protein per day must be taken in the diet to provide the minimum necessary for endogenous metabolism or it will be taken from the muscles. The foods high in protein particularly meat, fish, fowl, casein, milk, curd, gelatin, also nuts, chocolate and condiments as pepper and salt should be sharply restricted.

Foods to be limited also containing protein in considerable quantities are white flour, crackers, bread, toast, pastry, wheat cereals, Shredded Wheat, Cream of Wheat, cracked wheat, rice, farina, macaroni, corn meal, cornstarch, oatmeal, dried peas and dried beans.

Foods made up principally of carbohydrates, starches, sugars and cellulose, honey, cornstarch puddings or ice cream without eggs or gelatin or sherbets without egg white need little restriction. Dried fruits, apples, pears, peaches, nectarines, bananas, citrus fruits, grapefruit, oranges, lemons, grapes and cherries should be taken in as large quantities as possible.

In acute nephritis the question of a possible deficiency in important protective amino acids as methionine and choline must be considered and these supplied. It must be remembered that inflammatory exudates present in the renal tubules give rise to albumin and casts and the concentrating ability is less. Water should therefore be given in sufficient quantities to produce adequate output to rid the body of waste products even though it be dilute. Sodium intake should be very low.

Karo corn syrup solution is a good basis rather than milk which it must be remembered contains a moderate amount of protein. Fruit juices heavy with sugar may be given ad lib. Starvation of protein with excess of sugar intake keep nitrogen excretion at a very low level. Later on vegetables and cereals may be added bringing the protein intake up to 40 Gm. per day. Attempts should be made to maintain a nitrogen balance as soon as the nonprotein nitrogen shows no evidences of excessive retention.

In chronic nephritis protein intake must be maintained at a level that is just adequate yet at which there is no nitrogen retention. It is

## Chapter XXIX

### DOCK'S TWENTY DRUGS—PHYSICAL THERAPY

The firm conviction that proficiency in the use of a few good drugs was infinitely more valuable to the student and practitioner than a smattering of every item indexed in the old *Materia Medica* led Dr George Dock to select a list of twenty essential drugs. The Physician in Chief began in 1899 at the University of Michigan Hospital to encourage thorough study of, and practical familiarity with, this small list of drugs, each one the best of a class of drugs. There was no intention of limiting practice to twenty drugs but they were advanced as a necessary basis for further experiments in therapeutics.

Dock's twenty drugs are, for the most part, still in use fifty years after they were selected. Some have decreased much in importance and as the number 20 represented groups, each has been greatly expanded. Antibacterial sulfonamides and antibiotics as penicillin, streptomycin, and Chloromycetin have replaced many of the antisera, only a few of which remain. Vaccines have decreased in importance. Antihistamines, cholinergics, and sympathomimetics have been added, and insulin, liver extract, synthetic hormones, and vitamins have been appended as powerful pharmacodynamic agents to our therapeutic armament.

The twenty drugs are as follows: 1 Opium 2 Arsenic 3 Iodides and iodine 4 Iron 5 Quinine 6 Digitalis 7 Strychnia 8 Salicylates 9 Atropin 10 Bromides 11 Chloral veronal or trional 12 Ammonium chloride, balsams 13 Phenacetin 14 Calomel, sodium sulfate, castor oil, cascara 15 Bismuth 16 Nitrites 17 Acid hydrochloric, alkali, sodium bicarbonate 18 Ipecac 19 Male fern, santonium, thymol, or chenopodium 20 Sera, vaccines, opotherapy, thyroxin, etc.

Huchard and Fiessinger published a small book on the same subject of twenty drugs *La Therapeutique en Vingt Medicaments* Paris 1910, some years after lectures on "Twenty Drugs" were begun at Ann Arbor. The French authors listed the drugs as follows: 1 Sodium salicylate 2 Quinine 3 Mercury 4 Iodide 5 Digitalis 6 Iron 7 Sera 8 Collargol 9 Opotherapy 10 Bismuth 11 Theobromine 12 Sodium bicarbonate 13 Arsenic 14 Opium 15 Belladonna 16 Bromides 17 Castor oil, sodium sulfate or aloes 18 Nitrites 19 Ergot 20 Antipyrin.

#### General Rules

Whenever possible each drug should be given alone so that its effects can be more clearly observed. Close observation must be made for the

desired action It is better to begin with one or at the most two preparations of a drug In general drugs are not to be used to reduce temperature

### The Drugs

**Opium**—The derivative, morphine is still a sovereign remedy Codeine, Demerol and Methadon have been developed and have been found advantageous

**Arsenic**—New preparations as Mapharsen are still used but penicillin is at present the antisyphilitic drug of choice

**Iodide of potassium** lives on as a time honored alterative, organic iodides as Duodoquin etc are amebicides

**Iron**—Iron salts are effective and widely used in iron deficiency anemias

**Quinine** as an antimalarial has almost been displaced by chloroquine Quinidine has become indispensable in cardiac therapy

**Digitalis** powdered leaf has been retained in spite of the re introduction of crystalline digitoxin digilanids strophanthins Scillarin

**Strychnia** is obsolete Stimulants for various systems have been developed as analeptics for respiratory centers as nikethamide vago stimulants as Prostigmine sympathomimetics as Benzadrine sulfate

**Salicylates** are still the antirheumatic analgesics of choice Substitutes as amidopyrine and cinchophen are frequently toxic ACTH and Compound E may replace these

**Atropine** is retained as the vagoparalytic drug in spite of a host of tropic acid derivatives as Novatropine Syntropan Trasentine Pavatrine, etc

**Bromides** have been found toxic and accumulative and in spite of the anticonvulsive quality have been dropped in favor of one of the host of barbiturates

**Chloral hydrate** has been superseded by the barbiturates

**Ammonium chloride** has been retained not so much as an expectorant as an acidifier before giving diuretics Balsams are rarely used

**Phenacetin** is still used as an analgesic in mixtures but could be dispensed with

**Calomel** is rarely used as a cathartic, most saline cathartics are now avoided and cascara is obsolescent Hydrophilic gums of *Plantago ovado* are more natural laxatives

**Bismuth** is used now in the formal insoluble salicylate as an antisyphilitic but it is obsolescent as an antacid

**Nitrites**—Amyl nitrite and nitroglycerin are still the best prompt vasodilators for use in angina pectoris

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#### General Rules

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BAL, antiarsenic, antimercurial drugs are described in Chapter XXVII

Vasodilators and vascular alteratives as murex and niacinamide have widely displaced oral aminophylline and potassium iodide. Khellin is a new and promising drug.

Diuretics—Xanthines caffeine theobromine, and theophylline have been augmented and practically replaced by organic mercurials as Salyrgan, Theophylline, Mercuzanthin, Mercuhydrin, and Thiomerin.

**Acids**—HCl dilute is useful for replacement in patients with achlorhydria

**Antacids**— $\text{NaHCO}_3$  is used, but the carbonate or aluminum gels and magnesium trisilicate are antacids of choice

**Ipecac** is no longer needed as an emetic or as an amebicide. Emetine and Vioform, Chinofon, Diiodoquin and Chloroquine are the acceptable amebicides

**Anthelmintics**—Carbon tetrachloride has been replaced by tetrachlorethylene, hexylresorcinol, and gentian violet

**Sera and Vaccines**—Antisera are needed in some rare cases of pneumonia and meningitis, and in tetanus and diphtheria when the victims have not had toxin antitoxin or toxoid and booster doses. Agglutination test sera are needed in every modern laboratory

Vaccines against smallpox and typhoid fever are indispensable. Vaccines against viruses are being developed

**Opothrapy** has been greatly augmented by natural gland extracts and synthetic homologues. **Thyroid** extracts have been improved and standardized and thyroxin synthesized. **Epinephrine** (Adrenalin Suprarenin) has been synthesized and many homologues prepared as Neosynephrine, Paredrine, Benzedrine, Isuprel. **Ephedrine** of similar, more prolonged action even orally, has been developed from Ma Huang. **Insulin** extracts (of pancreatic islets) have been improved, concentrated, and mixed with protamine. **Liver** extracts contain the specific anti-anemia factor vitamin  $\text{B}_{12}$  for megaloblastic anemias

**Female sex hormones** (estrogens), theelin, prolan, luteinizing hormones, estradiol have been isolated and synthesized as have male sex hormones (androgens) as testosterone

**Adrenal cortex extract**, cortin (aqueous), lipo cortin (oil), adrenal corticosteroids have been identified as affecting salt and water, protein and carbohydrate metabolism

**Desoxycorticosterone** increases the salt and water retention, blood volume, and blood pressure

**Compound E** (Kendall) and **Cortisone** are antirheumatoid

**ACTH**, anterior lobe pituitary, adrenocorticotrophic hormone (Armour Laboratories) as antialarm reaction, antiallergic, antirheumatic, anti-fibrinoid, promises much. **Posterior lobe pituitary** extracts, Pituitrin, Pitressin, vasopressin, Pitocin and oxytocin (uterine contraction) are very effective and useful

**Hormones** are described at the end of the chapter on Endocrine Diseases

**Histamine antagonists** are described at the end of the chapter on Allergic Diseases

Other methods available are electric light cradles diathermy, micro waves and hyperthermia

### Actinic Radiation

Ultraviolet chemical or actinic rays are present in large quantities in sunlight at high altitudes where the air is free from the large quantities of solids found at a lower level, especially in the city. These rays are strongly bactericidal and exert a stimulating influence on the body metabolism. Actinotherapy has been used for some time in Switzerland, and has been adopted in the United States in the treatment of tuberculosis, skin disorders and general adjustment of metabolism.

It has been found that mercury vapor when heated to a high temperature by an electric current produces a light very rich in ultraviolet rays. This source of therapeutic light has been developed commercially in the form of so called quartz mercury lamps. Clinical and laboratory research has established the belief in medical quarters that this mode of therapy is a valuable adjunct in the treatment of bone tuberculosis especially bone and skin lesions septic wounds malnutrition rickets, and anemia.

### Massage and Exercise

The light stroking effleurage is useful locally in relieving reflexly pain and spasm especially in fractures and joint disorders. Generally, this type of massage has a soothing relaxing and sedative effect on the body as a whole. Deep stroking or drainage is employed for the purpose of reducing swelling and congestion in the extremities. Deep vigorous massaging is used locally for the purpose of building up the muscle cells especially in paralysis and other forms of muscle disease.

General exercise is given for body building or obesity. Corrective exercises are used for postural defects. Re educational gymnastics are of advantage in re establishing coordinated movements especially in paralysis and locomotor ataxia. Passive and late resistive movements are essential in convalescent heart cases especially after the establishment of compensation following an attack of heart failure.

## HYDROTHERAPY

### Scotch Douche

In this method of treatment streams of alternately hot and cold water are thrown from a rubber hose upon the patient at a distance of twelve feet under a pressure of 25 to 35 pounds.

**Action**—The effects depend upon the degree of variation of temperatures the extremes being  $49^{\circ}\text{C}$  to  $45^{\circ}\text{C}$  ( $120^{\circ}\text{F}$  to  $40^{\circ}\text{F}$ ). The



## PHYSICAL THERAPY

### (Action and Indications of Common Methods Used)

#### Electric Light Bath

A cabinet with 48 to 60 watts tungsten (Mazda) lamps is used in stead of the old fashioned Turkish bath, because it is more efficient, more sanitary, and safer. The patient and the treatment are always entirely under the control of the operator.

**Action**—The bath is either tonic, derivative, or eliminative, depending upon the temperature and the duration. Short exposure to intense heat is stimulating, while prolonged mild heat acts as a sedative. For diaphoresis, radiant heat with maximum illumination with a minimum degree of heat production is desirable, therefore, tungsten lamps should be used instead of carbon lamps. The light rays penetrate the tissues for some distance.

#### Indications—

1 Tonic. For patients convalescing from debilitating diseases or postoperative cases and in any case in which it seems desirable to build up the general vital resistance.

2 Derivative. To maintain a normal balance of the blood supply between skin and muscles on the one side, and the abdominal organs and pelvic organs on the other. It is the treatment par excellence for hypertension cases, and is also of value in venous stasis, heart disorders, especially with obesity, infectious arthritis, neuritis, and neurotic cases.

3 Eliminative. Wherever profuse sweating is indicated, as in uremia, chronic nephritis and gout cases, a cabinet bath is of service. The treatment is often used as preparatory to a Scotch douche.

#### Local Baking

Electric lighted devices containing lamps of from 250 to 1,500 watts for local application of radiant light and heat are used. The light rays penetrate the tissues to some extent and are there instantly converted into heat. This method is more effective than the older method of conductive heat which affected the tissues layer by layer.

**Action**—There is produced a hyperemia for the relief of deep seated congestion.

There is a relief of the local congestion by the hastening of the return circulation. The pain is relieved and an increase of leucocytes for combating local infections is produced.

**Indications**—Local hyperemia is used for local infections, inflamed joints, myositis, fractures, sprains, neuritis, and often as a preliminary to massage.

# PART V

## DIETETIC METHODS

### Chapter XXX

#### THE PREPARATION OF DIETS

Diet lists cannot be suitable for all patients with even the same metabolic disorders. The physician is best able to determine which and what amounts of foods are desirable and permissible for each patient. The Council on Foods and Nutrition of the American Medical Association discourages the listing of diets, yet many physicians need simple diet outlines as guides. The diets herein given represent the ideas of many physicians who have compiled diets of the types indicated. They are offered for whatever service and the saving of time they may accomplish with the expectation that each physician will modify the list as his experience indicates is desirable for his patients.

#### Requirements for Adequate Food Intake

The Basal Food Requirements for patients at rest in bed consist in sufficient carbohydrates and fats for basal total organ function burning or metabolism and enough protein to maintain nitrogen equilibrium, minerals and vitamins in amounts adequate to prevent deficiencies. The proteins yield glucose up to 54 % in the metabolic breakdown and supply four calories per gram while all of the starches and sugars furnish four calories per gram and the fats nine calories per gram. The basal calorie requirements depend upon age, sex, height and weight. Sitting about the room would add 10 % to the calorie requirements and walking or activity add another 10 % to the total calories.

#### Calculation of Diet Formulae

**General Routine Diets**—The patient at rest in bed requires on the average 30 calories per kilogram of the ideal body weight, a minimum of 2,100 calories, with much more needed if there be fever or hyperthyroidism. Enough calories must be provided to take care of the replacement of worn and torn tissues. The rebuilding and maintenance of nitrogen equilibrium is the function of the amino acids from proteins. Proteins should amount to 1 gram per kilogram per day. The ratio of proteins to carbohydrates should be 1 to 5 Gm. and proteins to fats 1 to 1.5 Gm.

pounding action of water under considerable pressure acts as a massage and enhances the reaction which is the aim of the treatment

**Indication**—A Scotch douche is always indicated following eliminative treatment to neutralize the depressing effect. The treatment is useful wherever an increased tone of muscular, circulatory, or nervous systems is desired. It is often of great benefit in sciatica, lumbago, myositis, arthritis, and in convalescent anemia cases.

### Whirlpool Bath

Water at a temperature of 40° to 45° C (105° to 120° F) is mechanically set into violent motion and the patient or the affected part is immersed.

**Action**—The effect is that of a stimulant to the blood and lymphatic circulations. It accelerates the retrogression of inflammatory processes and has a sedative effect in relieving pain and muscular spasm.

**Indications**—In Army practice the whirlpool was introduced for cases of gunshot wounds which had resulted in joint injuries, stiff and swollen joints, painful stumps, scar tissue, bone injuries, ankylosed and edematous limbs. The whirlpool bath is used in civilian practice with success in cases similar to those in which it was applied in the Army.

### Nauheim Baths

The original Nauheim baths of Bad Nauheim, Germany, are naturally impregnated with carbonic acid, sodium and calcium chlorides, and other salts of lesser importance. These baths have for many years been prepared in this country by charging artificially prepared brines with carbonic acid, either chemically or by means of mechanical devices.

**Action**—Through stimulation of the vasodilators of the peripheral or cutaneous circulation, blood is drawn from the congested visceral circulation and the heart is thereby relieved. With a relief of the venous stasis, the left ventricle is able to produce a more complete contraction and the pulse rate is reduced and becomes stronger and fuller.

**Indications**—Chronic cardiac, nephritic, hypertension, and neurasthenic cases usually improve under the Nauheim regime.

Other hydrotherapeutic measures available are continuous neutral sedative baths, hot and cold sitz baths, needle and shower baths, salt glow baths, hot and cold wet packs.

# PART V

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#### Calculation of Diet Formulae

**General Routine Diets**—The patient at rest in bed requires on the average 30 calories per kilogram of the ideal body weight, a minimum of 2000 calories, with much more needed if there be fever or hyperthyroidism. Enough calories must be provided to take care of the replacement of worn and torn tissues. The rebuilding and maintenance of nitrogen equilibrium is the function of the amino acids from proteins. Proteins should amount to 1 gram per kilogram per day. The ratio of proteins to carbohydrates should be 1 to 5 Gm. and proteins to fats 1 to 1.5 Gm.

Vitamins and minerals are obtained from raw fruits and vegetables which should make up one third of the diet

**High Caloric Diet** calls for 3 000 to 5,000 calories per day It is necessary to add fats in considerable amounts to make palatable high caloric diets Vegetable fats are preferable to animal fats

**Low Caloric Diet** may range from 2,000 calories down to 1 000 or even to 800 calories, made up mostly of vegetables and prescribed only in obese individuals

### General Instructions

- 1 Eat slowly and at the regular hours of serving
- 2 Sip liquids and masticate solids thoroughly
- 3 Take fluids moderately at meals, take water freely at other times
- 4 Avoid arguments and worrying, and rest after meals
- 5 Use fruits as laxative foods and drink two glasses of warm water each morning to prevent constipation, or take a hydrophilic gum of plantago as a natural laxative
- 6 Do not partake of a great variety of dishes at any one time or large quantities of anything very hot or very cold Avoid "frosted" drinks
- 7 Avoid undercooked vegetables overdone or hashed meats hard boiled eggs and any foods found to disagree

### Special Instructions

- 1 Take three meals daily and nothing between unless prescribed by the physician
- 2 No foods are permitted that are not on the diet
- 3 Foods or drinks brought in by visitors are withheld awaiting permission of the physician or dietitian for substitution

### Liquid Diet

Feedings every two hours from 2 A M to 8 P M and as nearly every two hours as possible when the patient wakes during the night Whole milk, buttermilk ice cream, cocoa, coffee tea strained soup fruit juice with water albumin water, meat juice gruels of cereals water and gelatin Serve 250 c c at each feeding

### Soft Diet

Three meals daily at 7 A M 12 noon, and 5 P M

At 10 A M, 3 P M, and 8 P M serve one article from liquid list

Anything on liquid diet may be served at mealtime

Margarine or fortified vegetable oil is preferable to butter

Cheese, cottage or Neufchâtel (American cream if finely grated)

Eggs according to taste (raw soft boiled, or poached)

Potatoes (white well cooked baked boiled creamed, scalloped with or without American cream cheese)

Bread (white or whole wheat) toast (do not remove crusts)

Crackers, Graham or soda (soft soaked in milk)

Fruits applesauce or baked apple (without skins) Other stewed fruits as apricots peaches prunes and canned fruits stewed until falling to pieces or run through strainer

Milk toast (made with skimmed milk or Borden Starlac)

Cereals flakes gruels mu h oatmeal rice macaroni Shredded Wheat biscuit grits Ralston's cereal Farina Cream of Wheat Malt O Meal (soft soaked in milk)

Puddings bread rice (made with raisins if so desired) chocolate meal, custard tapioca or Spanish or Bavarian cream

Ices or ice cream

Gelatin Jello or D Zerta (sugar free)

### Light Diet

Three meals daily at 7 A M, 12 noon and 5 P M

At 10 A M 3 P M and 8 P M select any one article from the liquid diet list

Anything on the liquid or soft diet lists may be served at mealtimes

Meats (prepare broiled baked or roasted once daily) scraped beef ground steak chopped stew veal or mutton chops poultry liver or kidney

One small serving of 4 ounces or 120 grams of any one meat or

Fish (no shell fish) (baked boiled or broiled)

Vegetables (well cooked) asparagus beans carrots celery, corn, peas spinach squash tomatoes turnips beets onions parsnips white and sweet potatoes (baked boiled mashed or creamed)

Fruit (fresh or canned) apples bananas (ripe) blackberries grape fruit cherries lemons oranges peaches raspberries strawberries water melon

Fruit (dried or stewed) apricots prunes figs raisins apples rhubarb

Bread brown graham corn rolls white zwieback

Crackers soda or Graham

Cream toast Hominy Cake

is thoroughly dissolved The powder contains fat 12 %, lactose 44 %, protein 34 %, and salts (minimum) 7 % One level teaspoonful will yield 14 cal One tablespoonful of Alacta to one ounce of water gives a mixture of the following values protein 40 %, fat 35 %, and sugar 55 %

*Skimmed Powdered Milk Solution, Starlac* Prepared by dissolving the milk powder in water (warm or at room temperature) and beating with fork until powder is thoroughly dissolved One tablespoonful of Starlac to two ounces of water gives a mixture which is approximately the strength of whole milk namely, protein 33 %, fat 35 %, and sugar 47 % Four tablespoonfuls of Starlac equals one ounce by weight, and furnishes 149 cal per ounce One tablespoonful equals 37.5 cal

*Powdered Milk, SMA* High in protein 37 %, average in fat 26 %, low in carbohydrate 30 %, and high in mineral 6 % Ca, P, and Fe

Follow the directions on the package for preparation One ounce yields 14 cal Add four packed tablespoonfuls to 330 cc water If half strength is desired, it may be diluted accordingly Other high, practically pure, protein solutions are Casec, Amigen, Protolysate or Protenum made up as per directions on the package

*Skimmed Milk or Protein Milk* Prepared as for whole milk using Borden's Starlac or with Mead's Protenum Low sodium milk is prepared from Lonolac according to the directions on the package These high protein powders may be mixed with skimmed milk and used as a beverage with the addition of sugar and flavoring substances

*Buttermilk Mixture, Lactic Acid Milk* Take a few tablespoonfuls of fat free lactic acid milk add 2½ level tablespoonfuls of flour to make a paste Make up to 1 quart with fat free lactic acid milk (1) Bring to a boil, withdraw from the fire and cool (2) Bring to a boil again and withdraw from the fire a second time (3) Add 4 level tablespoonfuls cane sugar, and bring to a boil for the third time (1, 2, and 3 should require about 20 minutes of time) Make up to 1 quart with boiled water if it has boiled away, place on ice

*Malt Soup* To 11 ounces (330 cc) of warm milk, gradually add 1¾ ounces (25 Gm) of flour stir constantly, then pour through a clean sieve or muslin In another dish dissolve 3 ounces by weight or 2½ tablespoonfuls of Borchardt's malt extract in 20 ounces (600 cc) of boiled water Then mix both solutions, put on the fire stir constantly and boil for 2 or 3 minutes

*Cream Soups* Cream soups may be made from vegetable pulp using 1 tablespoonful of cooked potatoes, peas or asparagus to ½ cup of water in which the vegetables were cooked ½ cup of sweet milk, and ½ teaspoonful of flour, with a little butter and salt Cook for another minute or two Strain, if necessary, and serve hot

**Vegetable Soup** One fourth pound of mutton stew cut into pieces 1 potato cut into pieces 1 carrot cut into pieces 2 stalks of celery cut into pieces 1 tablespoonful of pearl barley, 2 tablespoonfuls of rice 2 quarts of water Boil down to 1 quart, boil 3 hours Add pinch of salt and strain before feeding

**Lamb or Veal Broth** Lean meat chopped fine, 1 pound, cold water 1 quart, a pinch of salt, cook slowly 2 or 3 hours to 1 pint Add water from time to time so that when finished there will be 1 pint of broth Strain, and when cold skim off fat

**Chicken or Veal Broth or Consomme** Prepare a small chicken with all skin and fat removed or veal knuckle chop meat and if small cut bones and all into small pieces add 1 quart of boiling water and a little salt cover closely and allow to simmer over a low fire for 2 hours After removing allow to stand for 1 hour strain and cool remove congealed fat, serve or heat and strain. Add water from time to time so that there will be 1 pint when finished Veal oysters or clams may be substituted

**Beef Juice or Tea** Take  $\frac{1}{4}$  to  $\frac{1}{2}$  pound of round steak broil slightly, cut into small pieces and then press out the juice with a meat presser or potato ricer and add a small pinch of salt Do not heat sufficiently to coagulate albumin In making beef tea the beef cut in cubes is sealed into a small Mason glass jar and cooked by immersing in hot water which is kept simmering for 2 hours Serve fresh or warm before giving

**Soups** Meat or chicken stock may be enriched with purees cream, milk baby foods or proteolysates

**Purees** Creamed pea corn broccoli spinach asparagus celery (rubbed through a Foley mill or sieve)

**Potatoes** Boil potatoes in salt water in ordinary way until they are thoroughly done Then mash through a very fine sieve and add a little butter Bake select Idaho potatoes for serving in this form

**Spinach** Cook spinach in salt water until tender Pour cold water over it and drain Chop fine or rub through a coarse sieve To 2 tablespoonfuls of spinach add 1 teaspoonful of fine bread crumbs  $\frac{1}{2}$  teaspoonful of melted butter and a little salt Reheat and serve

**Pears** Soak 4 tablespoonfuls of beans and cook them slightly in a large quantity of water until they are soft but not broken Rub through a sieve add 1 cupful of soup stock and let them cook for  $\frac{1}{2}$  hour adding more stock if it boils away Mix in a little butter and flour about 1 teaspoonful of each and a little salt

**Asparagus** Make a pulp from the tender ends of some canned asparagus Make a sauce with  $\frac{1}{2}$  cup milk 1 teaspoonful of flour, and a little butter and salt Mix with 2 tablespoonfuls of the asparagus pulp



**Carrots** Cook  $\frac{1}{2}$  pound of carrots in a pint of salted water, adding more if it cooks away before the carrots are done. Rub through a sieve, add 1 teaspoonful of bread crumbs, a little butter and salt. Reheat and serve.

**Green Peas** Cook 1 cupful of green peas in boiling salted water until they are done. Drain, saving the water in which they were cooked. Rub through a coarse sieve. Make a sauce of 2 tablespoonfuls of water in which the peas were boiled, 2 tablespoonfuls of sweet milk,  $\frac{1}{2}$  teaspoonful of flour,  $\frac{1}{2}$  teaspoonful of fine bread crumbs. Mix all together. Reheat and serve. When canned peas are used, prepare as above after heating.

### Diets in Infectious Diseases

In the acute stage of infectious diseases, the diet should be liquid, and fluids should be forced to tolerance. Frequent small volumes, half glass, 125 cc, will be taken every half hour when a full glass, 250 cc, may be refused hourly. It must be remembered that in febrile conditions more fluids are needed because of greater losses by insensible perspiration and through the lungs. At least 2,500 to 3,000 cc are necessary in 24 hours. A febrile patient who gets adequate fluids will have a better chance to recover.

### High Caloric Typhoid Diets

**Directions for Nurses**—High caloric diet does not indicate the administration of a previously calculated number of calories. The caloric value is to be calculated at the end of each day. There must be individualization of feeding. Each patient, not the disease, must be fed.

Patients should be told that the more they eat, the sooner they will get well, and to ask the nurse for more food if they desire it. Each patient is to have as much food as he is capable of digesting. Note if there are any articles of food in the diet that the patient will not take or wishes changed. In such a case substitute articles of diet may be chosen from the approved list, if in accordance with the doctor's orders.

Eggs should be raw, coddled or soft boiled, never hard. They may be used in egg-nogs, custards, omelets, or scrambled.

All toast should be prepared with the crusts removed.

Unless otherwise specified, milk and cream mean whole milk and 20% cream. Twenty per cent cream is about the same as the top 4 ounces (120 cc) from a quart (1,000 cc) bottle of milk that has stood at least 6 hours. To add lactose to milk, boil 15 Gm ( $\frac{1}{2}$  ounce) in 30 cc (1 ounce) of water for 2 minutes, dissolve completely, cool, and add to milk.

To combine lactose, egg and coffee or tea Beat the egg lightly in a cup dissolve the lactose very thoroughly in very hot coffee or tea, pour onto the beaten egg stirring while pouring strain, add sugar or cream if desired and serve hot or cold

To make egg and lemon juice mixture Dissolve sugar in water, add lemon juice and pour onto beaten egg and add cracked ice or mix ingredients in a shaker and shake up thoroughly strain and serve If lactose is to be added, it is boiled with the sugar water for 2 minutes and cooled before adding lemon juice

All food is to be stopped immediately upon the appearance of any signs of hemorrhage or other gastro intestinal complications With vomiting all food is to be withheld for a few hours and then the diet reduced to its simplest form avoiding all rich mixtures

If tympanites is troublesome the excess of lactose must be removed secondly the eggs eliminated then the cream and lastly the total caloric intake reduced to 1 500 or 1 000 calories

Alcohol may be taken with advantage in the form of whisky (200 c.c. per ounce) (30 c.c.) by patients who persistently decline all food Or it can be used to increase the caloric intake on the low diets As much as 50 c.c. may be used every 3 hours

TABLE OF APPROXIMATE VALUES OF INGREDIENTS USED IN HIGH CALORIC DIETS

FOOD	AMOUNT	CALORIES
Applesauce without sugar	1 ounce (30 c.c.)	30
Apple pared and cored	1 medium about	75
Bread average slice	30 Gm.	80
Butter 1 pat	13 Gm. about	100
Cocoa, one rounding teaspoon	5 Gm	25
Crackers soda, 1 cracker	9 Gm	36
Crackers Uneda 1 cracker	6 Gm	25
Cream (10%)	1 ounce (30 c.c.)	60
Egg	1 average size	75
Egg white	white of one egg	15
Egg yolk	yolk of one egg	60
Farina (cooked)	1 heaping tablespoonful	25-30
Lactose (milk sugar)	1 heaping teaspoonful 10 Gm	40
Lactose (milk sugar)	1 measured ounce 18 Gm	72
Lemon Juice	1 ounce (30 c.c.)	12
Milk (whole)	1 ounce (30 c.c.)	20
Orange	1 large	100
Orange juice	1 ounce (30 c.c.)	15
Oatmeal (rolled oats cooked)	1 heaping tablespoonful	36
Potato	1 medium	100
Rice boiled	1 heaping tablespoonful, about	60
Sugar, cane	1 heaping teaspoonful	40
Sugar cane	1 level tablespoonful	56
Toast	average slice	80

## High Carbohydrate Typhoid Diet

### RECIPES AND SUBSTITUTES

1 Milk toast with toast and milk

2 Cocoa with milk

	AMOUNT	CALORIES
Cocoa	25 Gm	50
Lactose	50 Gm	240
Milk	135 c c	80
Cream	50 c c	120

Mix sugar and cocoa, cook in milk until dissolved    Serve with cream

3 Cocoa with cream

Cocoa	25 Gm	50
Lactose	50 Gm	240
Water	150 c c	0
Cream	100 c c	180

Mix cocoa and sugar, add water, and boil    Then add cream and serve with whipped cream

4 Coffee

<i>Strong coffee</i>	150 c c	0
Lactose	25 Gm	120
Cream	50 c c	120

5 Plain Junket or Rennet Custard

Lactose	25 Gm	100
Milk	150 c c	100
$\frac{1}{4}$ Junket tablet		
Water, cold	25 c c	
Vanilla		

6 Cocoa Junket

Cocoa	25 Gm	50
Lactose	25 Gm	100
Milk	150 c c	100
$\frac{1}{4}$ Junket tablet		
Cold Water	25 c c	

Mix cocoa and sugar add milk, and heat lukewarm stirring constantly while adding the dissolved Junket    Stir thoroughly and leave in a cool place to set

7 Soft Custard

Milk	250 c c	160
Egg	1	80
Lactose	50 Gm	240
Speck of salt		
Vanilla		
Caramel made from sugar		

Beat the egg slowly, add sugar, and beat in milk slowly    Cook in double boiler stirring constantly until it thickens a little    If it curdles, set in a dish of cold water and beat at once    Flavor and cool

## 8. Baked Custard

	AMOUNT	CALORIES
Lactose	40 Gm	160
Milk	150 c.c.	100
Egg	1	80
Speck of salt		
Nutmeg and vanilla		

Beat egg slowly, warm sugar and milk stir constantly add the egg strain in cup and flavor Bake in pan in moderate oven until knife comes out clean 30 to 60 minutes

## 9. Bread Pudding

Lactose	40 Gm.	160
Milk	150 c.c.	100
Egg	1	80
Bread	30 Gm.	60
Butter	1 Gm	1.0

Spread bread with butter and cut in squares Beat egg slightly beat into milk and sugar stirring constantly Pour over bread Grate on a little nutmeg and bake

## 10. Lemonade

Lactose	125 Gm	480
Cold water	200 c.c.	
Lemon juice	30 c.c.	

Boil sugar and water 5 minutes add lemon juice to taste strain, and cool

## 11. Vanilla Ice Cream

Cream (20%)	125 c.c.	350
Whole milk	60 c.c.	40
Lactose	60 Gm	240

Speck of salt

Flavoring

Syrup of fruit or chocolate may be substituted for vanilla This makes a total of 6 large tablespoonfuls

**Management**—Give nourishment every hour from 8 A.M. to 9 P.M. and on succeeding days additional feedings are to be taken on the hours indicated Push fluid intake to two three or four liters per 24 hours Do not discontinue an extra feeding that has been started on a previous day The amount of any food used as indicated in the first feeding thereof should remain constant

## Diet in Tuberculosis

The diet in tuberculosis is as important in the treatment of the disease as any other single factor Usually anorexia is a conspicuous feature of the disease and must be overcome before improvement or cure is possible

Essentially the important characteristics of the diet for tuberculosis patients are (1) The food should be simple easy of digestion and

tempting to the appetite (2) It should be high in calories—3,000 in average patients, more or less in certain cases (3) Protein in liberal quantities up to  $1\frac{1}{2}$  to 2 Gm per kilogram of body weight (4) A well balanced ration with an abundance of mineral salts, vitamins, and roughage, unless there is a complicating diarrhea

Individualization of the diet is a necessary requirement because patients with tuberculosis generally have digestive symptoms of greater or lesser severity. Patients who have intestinal as well as pulmonary tuberculosis must have a much more bland diet than would ordinarily be given.

For the average patient with normal digestion the use of the regular house diet, reinforced with supplemental feedings of milk and cream between meals and at bedtime, is sufficient.

In patients with more advanced tuberculosis, too much emphasis cannot be placed upon the manner in which the food is prepared and served. Many patients with moderately advanced tuberculosis suffer from marked anorexia and in them an attractive and well prepared tray may do much to overcome this disagreeable symptom.

Some patients with tuberculosis may show special digestive disturbances such as hyperchlorhydria or hypochlorhydria, achlorhydria, or diarrhea. The diets employed in those conditions, plus supplemental feedings to increase the calories, and supplemental dilute HCl and pepsin, are indicated.

### Sample Menu For Patients With Tuberculosis

#### *Breakfast*

Fruit, any kind  
Cooked or dry cereal with sugar and cream or milk thickened with Starlac  
Eggs, 2, any style except fried hard  
Bacon, 2 or 3 strips crisp  
Toast with butter 1 or 2 slices  
Coffee, tea or cocoa, if desired, with sugar and cream

#### *Midmorning Feeding*

8 oz. of whole milk plus 1 well beaten egg

#### *Dinner*

Soups are best omitted because they often tend to decrease the appetite for other foods and because they have low caloric value.

Meat, a liberal serving of any of the following: beef, lamb, chicken, turkey, veal, ham, fish or oysters, baked or broiled, are of major importance.

Potato, rice, macaroni, or spaghetti in average servings make up the caloric value.

Green vegetable salad of an average serving should be prepared as a source of vitamins.

Fruit salad, with or without gelatin mayonnaise, or French dressing may be substituted for fresh vegetables

Bread, crackers toast with butter

Desserts—any average serving of the following      usterds puddings, gela  
tin des erts ice cream plain cakes sponge cakes, ices, or if preferred,  
stewed canned or fresh fruits

Milk—a large glass

*Midafternoon Feeding*

Same as in morning

*Supper*

Eggs any style except fried hard

Meat—broiled ham or bacon

Pice or grits with butter

Bread and butter

Salad—lettuce and tomato or fruit salad with French dressing

Dessert—stewed fruit

Milk—a large glass

*Bedtime*

A large glass of milk

Such a diet will carry in excess of 3 000 calories and may be varied sufficiently to meet the tastes of the majority of tuberculosis patients. The high caloric diet and recipes described under Typhoid Diets may be used in tuberculosis patients

Proteolysates made from casein plasma, and liver may be added to crude mixtures of amino acids. Vegetable oils as olive oil Mazola, and soybean oil may be added instead of animal fats and as a substitute for dairy product fats

### The Dietetic Management in Heart Disease

The chief consideration in the dietetic care for patients with heart disease is usually concerned with the restriction of salt and the alleviation of edema. Edema is usually the result of a disturbed salt and water metabolism and the inability to excrete salt particularly contributes to the serious picture in heart failure. On the other hand, low blood serum protein levels occur at times and when there is a nephrotic element in the clinical picture and oligoproteinemia contributes to the edema formation. This is particularly likely to occur where there has been concomitant liver damage.

Consideration of food elements that might contribute to the development and improvement of heart muscle function as protein of high biological character amino acids choline and methionine, vitamins liver extract protein hydrolysates must not be neglected. The augmentation of protein and the elimination of fat are given in fatty liver cases more attention in the development of dietotherapy.

The evidence of particular importance of the sodium ion in the development of edema has been championed by Newburgh, and in recent years clinically proved by Schemm. He and others have demonstrated the value of low sodium, acid, and neutral ash diet in the upsetting of salt and water stores and in dissipation of brine from the body. The value of forcing fluids, rather than restricting fluids, in patients with edema has been advocated by Schemm. Schemm has pushed the fluid intake to higher levels than most physicians would go and many would consider somewhat dangerous. Some good points have been brought out in recent years, the dangers of water intoxication and of salt depletion are kept in mind even though they are only rarely precipitated complications.

The edema, salt, and fluid volume increase in the interstitial tissues. In composition it is made up of 10 Gm per kilogram or 1 000 cc of an alkaline mixture of 5 parts sodium chloride and 1 part of sodium bicarbonate at a pH of 7.4. This is subject to the vicissitudes of sodium deficiency or vaporization producing brine logged tissues and dehydrating the cells. Sodium is stored indefinitely retained, unless sodium bicarbonate is used up by ever forming acid salts of metabolism or ingestion of ammonium chloride.

Acidification incites kidney to balance the threat to pH concentration by elimination of neutral or sodium salts. As sodium leaves, water is free for diuresis producing weight loss, or for water vapor producing weight loss without diuresis, or to hydrate cells producing no weight loss or diuresis.

Retention and elimination of acid salts is physiological. There are slight fluctuations throughout 24 hours. Edema forms if changes continue indefinitely in one direction or if augmented. Dehydration results if continued at expense of body water. Water reaches the kidney only after all other demands for water are met and therefore the kidney's functional capacity is taxed in its ability to excrete a concentrated urine.

Reference Schemm F R Ann Int. Med 17 952-969, Nov 1942, 21 937-976,  
Dec 1944

### Karell Diet

This is the traditional dietetic treatment of edema—a low salt, low fluid, low caloric diet that is inadequate—too low in carbohydrates and relatively high in protein and in fat and, furthermore, yields an alkaline ash unless 4 slices of toast are added.

A total of 800 cc of milk divided into four feedings of 200 cc each are to be given at 4 hour intervals during the day, 8 A M, 12 noon 4 P M, and 8 P M.

It is used chiefly as a temporary dietetic treatment in congestive failure, acute nephritis and chronic nephritis with edema. When so used, it should be continued for not more than five to seven days, after which time the modified Karell Diet may be substituted as follows:

Eighth Day	Milk as above 10 A.M.—1 soft boiled egg 6 P.M.—2 pieces of dry toast
Ninth Day	Milk as above 10 A.M.—1 soft boiled egg and 2 pieces of dry toast 6 P.M.—1 egg and 2 pieces of dry toast
Tenth Day	Milk as above 12 Noon—poached egg on toast, rice boiled in milk, vegetables 6 P.M.—1 soft boiled egg
Eleventh to Twelfth Days	Same as Tenth Day

No fluids, not even water, allowed except the amounts contained in the diet. No salt is used. Salt-free toast and butter used. Small amounts of cracked ice allowed with diet. Lonalac 280 grams in 2,000 c.c. of distilled water with sugar added and 500 c.c. of orange juice makes a 50 mg.  $\frac{1}{2}$  diet.

### Modified Rice Diet

Five ounces of uncooked rice is the usual daily portion unless otherwise specified. This is to be divided and used for two meals. White, brown or wild rice may be used.

*Methods of preparation.* One cup of uncooked rice daily is used (more if desired). Place about one quart of water in a pan and bring to a boil, add rice which has been washed to the boiling water. Add no salt. Allow to boil 15 minutes or until the grains are tender. Next drain rice through a strainer and pour boiling water over it. Allow to drain. This amount makes three average servings. Brown rice requires longer cooking—about 25 or 30 minutes. After it is once cooked the rice may be steamed or baked with any fruit desired. White or brown sugar may be used as much as desired. Add no butter, margarine, drippings, lard, grease, salad oil, etc.

The third meal must include one food to be selected from list below.

Diazal (E. Fougere & Co.) may be used for seasoning instead of table salt.

Cereal	Quantity
Sweet potato (baked)	1 large
Irish potato (baked)	1 large
Palsan Wheat	$2\frac{1}{2}$ oz. (uncooked)
Spaghetti	2 oz. (uncooked)



<i>Cereal</i>	<i>Quantity</i>
Cracked Wheat	2½ oz (uncooked)
Wheatena	2¼ oz (uncooked)
Wheat (shredded biscuits)	2 biscuits
Wheaties	2¼ cups
Macaroni	2 oz (uncooked)
Sparkies Rice (Quaker)	3 cups
Sparkies Wheat (Quaker)	3 cups
Corn meal (yellow or white)	2 oz
Cream of Wheat (5 minutes)	2 oz
Farina (Quaker)	2½ oz
Hominy grits	3 oz
Salt free bread	3 slices

Milk, four to six ounces Lonalac (Mead's milk may be used on cereal) Two heaping teaspoonfuls of powdered milk to six ounces of water This is easily mixed in a "whirl mixer"

Three ounces of lean meat daily All fat must be removed before cooking Hamburger, lamb, chicken, roast beef or liver may be used

Two vegetables from the vegetable list daily

Chard	1 cup	Broccoli	1 cup	Asparagus	1 cup
Spinach	1 cup	Cauliflower	1 cup	Onions	½ cup
Beets	¼ cup	Cabbage	1 cup	Cucumbers	1 cup
Carrots	½ cup	Lettuce	1 cup	Squash (with out seeds)	½ cup
Turnips	1 cup	String beans	½ cup		
Escarole	1 cup	Tomato	2 cups		

Fresh, canned, and dry fruits from fruit list Diet should include 10 to 12 servings daily of the following

#### *Fruits Approved*

Fresh fruits	Cranberries	Plums
Frozen fruits	Whole figs in heavy syrup	Peaches
Canned fruits (free of preservative)	Grapefruit	Pears
Stewed dried apricots	Pure honey	Pineapple
Berries of all kinds, fresh or frozen	Jellies and jam—if pure	Dried prunes
Bananas	Lemons	Raisins (boiled to soften if eaten on rice)
Cherries (black Bing or Royal Ann)	Melons	Tangerines
	Oranges	

Liquids should include only one quart fruit juice daily No vegetable juice, no tomato juice, and no juices containing preservative (sodium benzoate)

The following may be used as desired White sugar brown sugar, Karo, maple sugar and maple syrup jelly gumdrops chocolate sauce (not over 1 oz) and marshmallow whip (2 tablespoonfuls)

Fats should be avoided when oils are needed use only mineral oil Permit No molasses No dates No dried figs No nuts No maraschino cherries No avocados No canned fruits or fruit cocktail which contains maraschino cherries No spices No table salt or baking soda or other antacid mixtures containing sodium

Meat may be pan fried in a mixture of mineral oil and lemon juices adding some cooked rice to the gravy in the pan with a little water and browned slightly makes a good change

Meat may be ground and mixed with cooked rice out of which may be made a hash or mold into cakes and then cook in mineral oil and lemon juice The mixture may be baked with tomatoes seasoned with onion or a little garlic

Meat may be placed on top of uncooked rice and cooked in a pressure cooker following the directions for boiling rice in a pressure cooker

For charcoal broiling of steak or chops or other meat a tin can with holes punched on the sides near the bottom and half filled with charcoal makes a small broiler Place steel skewers through the meat and suspend over the top of the can This method of cooking improves the taste of unseasoned meat

Diasal (E Fougera & Co) may be used for seasoning instead of table salt

Hominy grits may be cooked and allowed to cool in deep pan The cold grits may be sliced in  $\frac{1}{2}$  inch strips and fried in mineral oil and eaten while hot with maple syrup or honey

*Essential additions* Ferrous sulfate 0.25 Gm 3 times daily Unicaps or essential vitamin additions 2 or 3 times daily

### Neutral Low Sodium Diet Treatment of Edema

The patient is weighed on a weighing stretcher on admission and daily before breakfast if possible and the weight recorded

The fluid intake and the urine output from 7 A.M. to 7 A.M. daily should be recorded

Continue the patient's cardiac tonic therapy as maintenance doses of digitalis 0.1 Gm ( $1\frac{1}{4}$  gr) daily Ammonium chloride 6 Gm (90 gr) and Thimeron are given as needed

In the presence of massive anasarca with embarrassing hydrothorax ascites or extreme congestion liver engorgement or pulmonary edema more or less heroic emergency measures as paracentesis or venesection may be necessary

**A Modified Karell Diet** 6 cups Lonalac with 4 slices of bread or 2 cups of cereal or rice in six small feedings with no salt substitute added

or used in the preparation of the food. No extra milk, fruit juices or bouillon should be allowed. This is a neutral diet that is low in salt, low in sodium, and low in mineral ash. The bread, cereal, or rice are acid ash foods to balance against milk and cream, basic ash foods.

If later on vegetables and fruits or their juices are added, the resulting alkalinity must be neutralized by acid foods as prunes, plums, cranberry, fowl, fish, or meat.

Bread, butter, rice, or cereal must be unsalted.

Gelatin powder 1 tablespoonful (10 Gm.) may be given tid.

Distilled water with two drops of diluted HCl per glass should be given to tolerance.

Glucose in 5 % solution may be given in 500 cc. to 1,000 cc., introduced very slowly, 30 cc. per hour into the sternal or iliac bone marrow.

When the acute and threatening symptoms abate and foods and fluids are taken freely, the diet may be increased but care must be taken to maintain a neutral or acid ash base.

The fluid intake should be increased to at least 2,500 cc. by giving glasses of distilled water to which 5 drops of dilute HCl has been added every hour throughout the day. Clear tea, diluted prune, plum or cranberry juices, and unsalted beef or chicken broth may be used.

If necessary to maintain the intake at 2,500 cc. glucose 5 % solution may be given intravenously in an amount up to 1,000 cc.

No alkalis should be used to combat distention.

High potassium diet calls for either 2 pounds potatoes or 2 pounds of bananas ( $\frac{1}{2}$  pound servings four times a day—two servings of potatoes and two of bananas may be used in place of the four servings of either one). Hungarian paprika used on the foods adds much potassium. Either potatoes or bananas or half of each may be used in place of 800 cc. milk of the regular Karell diet.

### Basic Rice Diet for Patients Who Are Hospitalized Under Constant Control of Blood and Urine Chemistry

**Rice** Any kind, polished, brown, or wild rice. Boiled or steamed without salt, milk, or fat of any kind (no butter, margarine, drippings, lard, grease or salad oil).

**Fruit** All kinds, preferably fresh (raw or stewed), but also dried, frozen, canned or preserved. (No salt added.) No nuts, dates or avocados.

**Sugar** White or brown. Honey but no syrups. (No Karo, etc.)

**Fruit Juices** All kinds, fresh or canned, without sodium benzoate. (No vegetable juices, no tomato juice.)

**Fluids** (Only fruit juices or distilled water) Limited to about  $1\frac{1}{2}$  to 2 pints daily.

## Strict Limitation of Sodium Intake and Forced Fluids in the Treatment of Edema

The importance of the sodium ion in the tissue or interstitial fluids in the maintenance of edema has been established by Newburgh and his assistants. The older idea of the hydrophilic properties of the chloride ion has been set aside. It had been common practice for many years to prescribe a low salt low protein diet for patients with edema. Keith and his group later introduced the use of acid producing salts, ammonium and potassium chloride or nitrate to aid in the mobilization and elimination of sodium by producing a tendency to tissue acidosis in edematous patients. Schemm has re-emphasized the importance of the sodium ion in edema and he has used a diet in which the sodium was limited sharply as possible and the ash was neutral or acid. In addition he forced fluids to at least four and often six liters per day even in the presence of congestive heart failure to wash the sodium from the tissues. He reported spectacular results and concluded that the sodium ion was more important apparently than the venous stasis.

### Low Sodium Diet

The complete elimination of all sodium from the diet is impossible since most natural foods as eggs milk butter cheese, and meat have a fairly high inherent sodium content.

The purpose of the diet is to reduce the amount of sodium eaten to as low a level as is practical and at the same time to allow sufficient amount of food to maintain a constant weight.

Sodium is present in many substances of which common table salt (sodium chloride) baking soda (sodium bicarbonate) and food preservative (sodium benzoate) are the most frequently used rich sources.

In view of the fact that innumerable medicines (Alka Seltzer and acids laxatives sedatives as bromides etc) contain sodium it is wise to allow no medicine unless prescribed by the physician. One small dose of a medicine may contain more sodium than would be included in several days of the diet.

All substances contain some sodium therefore even a diet composed of foods whose sodium content is relatively low must be restricted in quantity. Thus if an excess of the foods in this diet is eaten, the amount of sodium will be increased.

Foods for the day's diet are to be selected from those listed below. If each item is carefully weighed or measured the diet will contain ap

or used in the preparation of the food. No extra milk, fruit juices, or bouillon should be allowed. This is a neutral diet that is low in salt, low in sodium, and low in mineral ash. The bread, cereal or rice are acid ash foods to balance against milk and cream, basic ash foods.

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In view of the fact that innumerable medicines (Alka Seltzer, antacids laxatives sedatives as bromides etc) contain sodium it is wise to allow no medicine unless prescribed by the physician. One small dose of a medicine may contain more sodium than would be included in several days of the diet.

All substances contain some sodium therefore even a diet composed of foods whose sodium content is relatively low must be restricted in quantity. Thus if an excess of the foods in this diet is eaten, the amount of sodium will be increased.

Foods for the day's diet are to be selected from those listed below. If each item is carefully weighed or measured the diet will contain ap

proximately 200 mg of sodium. No salt, soda or baking powder is to be used in the preparation of any food, and no salt is to be added at the table. Success may rest in the reduction of sodium intake from 1 Gm to 250 mg per 24 hours. *Salt free bread* may be gotten from certain bakeries. Passover bread and Matzoth may be used.

*Dialyzed milk* only is included in very low sodium diets, because milk contains 50 mg % of sodium. Obviously, a single glass of milk contains 100 mg of sodium. *Mead Johnson's Ionalac* (powdered low sodium milk) may be used. A *protein hydrolysate* as *Lactamin* is low in sodium and each ounce contains 12 Gm of nitrogen equal to 75 Gm of protein. Diluted with water the solution tastes badly but may be given as medicine.

The diet, without milk and sugar, contains about 1,300 cal. Since neither of these foods contain sodium, they should be added in amounts to obtain the desired calories for the day's diet. The amount of these substances that can be included from a practical point of view are limited. It is not feasible to include more than 8 tablespoonfuls of margarine and 4 tablespoonfuls of sugar. If this amount is included, the diet will contain 2,500 cal.

*Meats* furnish essential protein of high biological value. 150 Gm (5 ounces) of any one of the following

Beef steak, 3' x 2' x 1', Roast, 3½' x 2" x 1½"	Venison
Chicken breast, 3¼' x 2' x 1'	Sweetbreads
Turkey breast, 3¼' x 2' x 1'	Fish, fresh water
Lamb, chop or roast, 3½' x 2' x ½'	Bass
Mutton, chop or roast, 3½' x 2" x ½"	Trout
Pork roast, lean, 3½' x 2' x ¼'	Perch
Veal cutlet or roast, 3½" x 2' x ½'	Crappie
Squirrel	Catfish, fresh water

Egg albumin is good protein, but yolk, due to its relatively high sodium and fat content cannot replace meat.

Ordinary commercial bread contains approximately 100 mg of sodium per average slice. Care must be taken to be certain that no salt has been included in preparation of the bread used on this diet.

Bread may be omitted entirely and instead ½ cup of unsalted cooked macaroni, spaghetti, rice, or farina may be used in place of one slice of bread. Since the diet does not include milk or cream, butter may be used with these foods.

Butter or margarine must be sweet or have all salt washed out of it.

Potato (peeled and boiled or baked) and Cereals are satisfactory in amounts of  $\frac{1}{2}$  cup of any one of the following macaroni spaghetti, rice, farina cornstarch puffed rice, rolled oats Shredded or winter wheat or germ barley, but

NOT corn flakes grapenuts, dried beans navy, lima, peas or cow peas

These are the same articles that may be substituted for bread If the two slices of bread are omitted, the allowance in this group will be increased by one cup This would permit for example  $\frac{1}{2}$  cup of each of the following potato macaroni, and rice If one slice of bread is omitted the allowance in this group will be two  $\frac{1}{2}$  cup servings

Vegetables are most desirable in amounts of  $\frac{1}{2}$  cup of any one of the following

I Asparagus tips cucumbers eggplant squash, pumpkin, onions peas (green)

II Green beans broccoli Brussels sprouts, cabbage, cauliflower, endive, lettuce mushrooms tomatoes

III Parsnips green peppers okra carrots ( $\frac{1}{4}$  cup only) salsify beets kale

Commercially canned vegetables cannot be used on this diet due to the salt and sodium benzoate that has been added in preparation Vegetable oils vinegar pepper and paprika, and Neocurtasal may be used in small amounts as seasoning and in salad dressings No mayonnaise catsup chili soya sauces and no dried or canned fruits as figs or raisins should be added

Fruits—Fresh frozen stewed or baked, or as juices are prescribed in  $\frac{1}{2}$  cup serving of any three of the following

I Blackberries cherries fresh figs loganberries pears plums raspberries

II Apples apricots gooseberries grapes oranges or juice lemons peaches blueberries, tangerines, grapefruit mulberries

III Bananas cranberries plums quince pineapple, rhubarb, water melon strawberries prunes 3 or 4 No cantaloupe

Nuts absolutely unsalted or unblanched Almonds Brazil pecans and peanuts

Sugar—Honey chocolate or jelly clear, in 3 tablespoonful servings are acceptable as desserts

Milk 1 pint 500 cc only dialyzed or made of Lonolac Powder (Mead Johnson and Co) per day may replace peanuts and butter to maintain the protein at 70 Gm per day



*Cream* 20 %— $\frac{1}{4}$  cup should be omitted in low fat diet

*Coffee* or tea or cocoa (not Dutch Process) as desired No soda pop or soft drinks *Distilled water*, ad lib Ammonium, calcium, magnesium, potassium or betaine chloride, Diasal, and Neocurtasal may be used as a salt substitute

### Sample Menu

(Approximately 0.7 Gm of sodium and 2,000 total calories)

#### Breakfast

1 fruit from Group II  
1 2 slices of salt free bread  
1 pat sweet butter  
1 egg  
Coffee or tea with 1 oz  
cream

#### Lunch

1 serving meat  
1 serving cereal product or  
1 potato  
1 vegetable from Group I  
1 vegetable from Group III  
1 2 slices salt free bread  
1 serving fruit from Group  
I  
1 glass milk

#### Dinner

1 serving meat  
1 potato  
1 vegetable from Group I  
1 vegetable from Group II  
1 2 slices salt free bread  
1 pat special butter  
1 fruit from Group II  
Coffee or tea with 1 oz  
cream

Eat no more than described in the diet

### References

- Wheeler, E O Bridges, W C, and White P D Diet Low in Salt (Sodium) in Congestive Heart Failure *J A M A* 133 16, 1947  
Schroeder H A Studies on Congestive Heart Failure *Am Heart J* 22 141, 1941  
Schemm, F R High Fluid Intake in Management of Edema Especially Cardiac Edema II Clinical Observations and Data, *Ann Int Med* 21 937, 1944  
Kuh, Joseph R A Practical Low Sodium Diet *Permanente Found M Bull* 5 130 135, 1947

### General Cardiac Diet

During severe cardiac decompensation the diet should be liquid and gradually increased to a soft diet The fluid intake should be restricted to from 2,000 to 2,500 cc depending upon the amount of edema present The diet should be salt free The Karell diet is temporarily used with satisfactory results On the third or fourth day such additional food as toast, crackers, jelly stick candy, etc may be prescribed About the eighth day add strained fruit and additional vegetable purées

The diet should provide for normal metabolic requirements and contain feedings of such volume and consistency as to call for minimum effort in ingestion and least distress subsequently

Adequate protein content is 0.7 Gm to 1 Gm per kilogram theoretical body weight Dry cottage cheese provides good protein and methionine, powdered gelatin supplies glycine

For additional energy add carbohydrates unless contraindicated  
For decreased energy reduce fat in diet

To relieve diarrhea or gas formation reduce carbohydrates and roughage temporarily

## RECOMMENDED FOODS

## For liquid diet

Gruel (well cooked thin, watery)  
Fruit juices (well strained)  
Desserts custards ices ice cream g latin  
Milk and e g whites whipped together  
Soups clear broth, beef tea soups (well strained)  
Beverages skimmed milk weak tea and weak coffee

## For soft diet all foods above and

Cereal (well cooked)  
Spaghetti and macaroni (plain cooked)  
White bread (not fresh)  
Soda crackers  
Pureed vegetables  
Baked potatoes  
Mashed potatoes

Cooked fruits (without seeds coarse skins or heavy fiber)  
Simple desserts custards sponge cake, angel food  
Eggs (soft cooked)  
Cottage cheese  
Oleomargarine

## For light diet all foods above and

Prepared cereals  
Graham bread or crackers  
Young vegetables (not coarse)  
Cooked fruit's  
Citrus fruits

Simple cakes  
Cheese (creamed or grated)  
Poultry  
Fish  
Sweetbreads

## Low Caloric Cardiac Diet I 356 Calories

FOOD	HOUSEHOLD MEASURE	TOTAL AMOUNT GRAMS	GRAMS					
			CARB	PROT	FAT	Ca	P	Fe
Skimmed milk	1 cup	240	12	8	1	293	930	0000
Meat mod. fat	3 1/4 x 2 1/2 x 3/8	40		10	3	006	112	0015
Cottage cheese	7 tea spoons	30	1	7		088	985	
Bread whole grain	1 slice	30	16	3		015	060	0007
Vegetables 5 %	2 sauc e dishes	200	8	2		072	002	0008
Fruit 10 %	1 serving	100	11			045	021	0000
Total grams			48	30	4	469	760	0041

## THE DIET I PRESCRIPTION GIVEN TO THE PATIENT

## FOOD FOR THE DAY

Skimmed milk one cup  
Meat 1 1/4 ounces  
Cottage cheese 4 level teaspoonfuls  
Bread whole grain 1 slice  
Vegetables 5 % 2 servings

## MEAL PLAN

## Morning

4 level teaspoonfuls cottage cheese  
1/2 slice bread  
1/2 cup skimmed milk  
Tea or coffee

Fruit, "10 %" 1 serving

These foods or their equivalents can be combined into "made dishes"

No concentrated sweets allowed, such as sugar, jelly, candy, cream, pastries, or cake

Fluid restricted to 1 quart (4 cups) daily, including milk, coffee, tea, clear broth, and water

Noon

1½ ounces meat  
1 serving vegetable  
¼ cup skimmed milk  
1 serving fruit  
Tea or coffee

Night

3 level teaspoonfuls cottage cheese  
1 serving vegetable  
½ slice bread  
¼ cup skimmed milk  
Tea or coffee

### Low Caloric Cardiac Diet II 609 Calories

FOOD	HOUSEHOLD MEASURE	TOTAL AMOUNT GRAMS	GRAMS					
			CARB	PROT	FAT	Ca	P	Fe
Skimmed milk	1 cup	240	13	8	1	293	230	0008
Meat, mod fat	3½ × 2½ × ½ inch	60		14	7	008	150	0020
Egg	one	50		6	5	033	090	0015
Cottage cheese	3 tablespoons	45	3	9		112	366	
Bread, whole grain	1½ slices	45	24	4		022	098	0011
Vegetables, "5 %"	2 sauce dishes	200	8	2		022	052	0008
Fruit, "10 %"	3 servings	300	33			135	063	0015
Total grams			60	43	13	625	1049	0015

### THE DIET PRESCRIPTION GIVEN TO THE PATIENT

#### FOOD FOR THE DAY

Skimmed milk 1 cup

Meat, 2 ounces

Egg 1

Cottage cheese, 3 level tablespoonfuls

Bread, whole grain, 1½ slices

Vegetables, "5 %" 2 servings

Fruit, "10 %" 3 servings

These foods or their equivalents can be combined into "made dishes"

No concentrated sweets allowed such as cake, sugar, jelly, candy, cream, pastries

Fluid restricted to 1 quart (4 cups) daily, including milk, coffee, tea, clear broth, and water

#### MEAL PLAN

Morning

1 serving fruit  
1 level tablespoonful cottage cheese  
½ slice bread  
¼ cup skimmed milk  
Tea or coffee

Noon

2 ounces meat  
1 serving vegetable  
1 level tablespoonful cottage cheese  
½ slice bread  
¼ cup skimmed milk  
1 serving fruit  
Tea or coffee

Night

1 egg  
1 serving vegetable  
1 level tablespoonful cottage cheese  
½ slice bread  
¼ cup skimmed milk  
1 serving fruit  
Tea or coffee

## Low Caloric Cardiac Diet III 1,100 Calories

FOOD	HOUSEHOLD MEASURE	TOTAL AMOUNT GRAMS	GRAMS					
			CARB	PROT	FAT	Ca	P	Fe
Milk whole	1½ cups	360	18	12	15	43	334	0009
Meat mod. fat	3½ × 2½ × 1	120		27	14	016	300	0040
Egg	one	50		6	5	033	090	0015
Bread, whole grain	3 slices	90	48	9		045	196	0021
Vegetables 5% "	3 sauce dishes	300	12	3		033	078	0012
Fruit, 10% "	3 servings	300	33			13	063	001
Butter	1 tablespoon	15			13	003	003	
Total grams			111	57	4	696	1 064	011

## THE DIET PRESCRIPTION GIVEN TO THE PATIENT

## FOOD FOR THE DAY

Whole milk, 1½ cups  
 Meat, 4 ounces  
 Egg 1  
 Bread whole grain 3 slices  
 Vegetables 5% 3 servings  
 Fruit 10% 3 servings  
 Butter 1 level tablespoonful  
 These foods or their equivalents can be combined into 'made' dishes  
 No concentrated sweets allowed such as sugar, jelly, candy, cream pastries, cake  
 Fluid restricted to 1½ quarts (6 cups) daily including milk, coffee, tea, clear broth and water

## MEAL PLAN

## Morning

1 serving of fruit  
 1 teaspoonful butter  
 1 slice bread  
 ½ cup milk  
 Tea or coffee

## Noon

4 ounces meat or fish  
 1 serving vegetables  
 1 teaspoonful butter  
 1 slice bread  
 ½ cup milk  
 1 serving fruit  
 Tea or coffee

## Night

1 egg  
 2 servings vegetables  
 1 teaspoonful butter  
 1 slice bread  
 ½ cup milk  
 1 serving fruit  
 Tea or coffee

## High Carbohydrate Cardiac Diet

Provide sufficient energy in an assimilable form of carbohydrates and milk, ripe bananas or mashed potatoes, any vegetables, purées, cream, butter, eggs, cooked cereals, fruit juice with sucrose, lactose, dextrose or dextrin, maltose and 1500 cc fluid. Give in small frequent feedings to avoid gastric retention, fermentation and distention.

Soft to light diet restricted in fluids and salts can then be gradually added. After the first week or on the third or fourth day, add toast, crackers, jelly or candy.

Insufficient nourishment may be in some instances responsible for poor response to digitalis.

A formula of 50 P 250 CH, 100 I with 2,000 calories for the first week with gradual additions to as high as 3,000 calories after the second week. The formula should be distributed over 5 or preferably 7 feedings as follows:

*Breakfast at 7 A M or 8 A M*

Cereal (cooked) 120 Gm  $\frac{1}{4}$  cup  
Milk 100 Gm  $\frac{1}{2}$  cup  
Cream 66 Gm  $\frac{1}{4}$  cup

{ Sucrose 10 Gm 3 teaspoonfuls  
and  
Glucose 10 Gm 3 teaspoonfuls  
and  
Dextrin maltose 10 teaspoonfuls  
or  
Sucrose 30 Gm 2 tablespoonfuls

*Dinner 12 Noon*

Soup { Cream 130 Gm  $\frac{1}{2}$  cup  
Potato 50 Gm  $\frac{1}{4}$  cup  
Butter 10 Gm 2 teaspoonfuls

Ice cream or Junket 100 Gm  $\frac{1}{4}$  cup

Milk 100 Gm  $\frac{1}{2}$  cup

Water 100 Gm  $\frac{1}{4}$  cup

or

Crushed ice 1 cup

*Supper 5 P M*

Soup, puréed vegetables,

Spinach 50 Gm  $\frac{1}{4}$  cup

Milk 100 Gm  $\frac{1}{2}$  cup

Cream 66 Gm  $\frac{1}{4}$  cup

Butter 10 Gm 2 teaspoonfuls

{ Egg 50 Gm  
Milk 100 Gm  $\frac{1}{4}$  cup  
Lactose 10 Gm 3 teaspoonfuls  
and  
Custard { Glucose 10 Gm 3 teaspoonfuls  
or  
Sucrose 20 Gm 1  $\frac{1}{2}$  tablespoonfuls

*Lunches 9 A M*

Crushed ice or water 100 c c 1 cup or  $\frac{1}{2}$  cup

*10 A M*

Orange juice 150 c c  $\frac{3}{4}$  cup

Lemon juice 50 c c 1 teaspoonful

{ Sucrose 10 Gm 3 teaspoonfuls  
and

Glucose 20 Gm 6 teaspoonfuls

or

Sucrose 30 Gm 2 tablespoonfuls

*3 P M*

Milk 100 Gm  $\frac{1}{2}$  cup

Cream 66 Gm  $\frac{1}{4}$  cup

{ Dextrin maltose 12 Gm 4 teaspoonfuls  
and

Sucrose 6 Gm 1 teaspoonful flavoring

or

Sucrose 1 tablespoonful

*4 P M*

Water 100 c c  $\frac{1}{4}$  cup

or

Crushed ice 1 cup

*7 P M*

Milk 150 c c  $\frac{3}{4}$  cup

{ Dextrin maltose 10 Gm 3 teaspoonfuls  
and

Sucrose 5 Gm 1 teaspoonful

or

Candy 1 stick

**References**

- Kempner W Compensation of Renal Metabolic Dysfunction Treatment of Kidney Disease and Hypertensive Vascular Disease With Rice Diet, North Carolina M J 6 61 (Feb) 117 (Mar), 1945  
Grollman A and others Sodium Restriction in the Diet for Hypertension J A M A 129 533, Oct, 1945  
Dick, G F, and Schwartz W B Response of Experimental Hypertension to a Rice and Fruit Juice Diet, Proc Soc Exper Biol & Med 65 22, May 1947  
Flipse M E, and Flipse M J Observations in the Treatment of Hypertension With Rice Fruit Diet South M J 40 721, Sept, 1947  
Editorial Postgraduate Medicine 2 211 Sept 1947

### Peptic Ulcer Diets

The dietary management of peptic ulcer constitutes one of the most important measures in treatment. The principles to be employed providing ulcer diets are simple. The diet should be nonirritating, bland, and smooth. It should be composed of foods which do not remain in the stomach for a long time and should be easily digestible. The feedings should be given in small amounts at frequent intervals. Sufficient variation should be accomplished to prevent monotony in the diet since it has to be continued for long periods.

The dietary treatment usually recommended is a modification of the original Sippy, or the Mills, Meulengracht, Andresen or a Hodges, Machella and Miller Protolysate diet.

In the Sippy regime upon admission to the hospital the patient is given 3 oz. of an equal part milk and cream mixture each hour on the hour from 7 A. M. to 7 P. M. If obese or adverse to cream use skimmed milk thickened with powdered whole or skimmed milk. This is continued for from 2 to 5 days or more or until the patient has relief from pain vomiting or bleeding.

On the second to the fifth day there may be added to the diet 3 oz. of a well cooked cereal such as Cream of Wheat or strained oatmeal. This may be given at the two feedings corresponding to breakfast and supper.

The following day a soft boiled egg may be given with the midday feeding and a day later thin hard toast (Melba toast) may be given at each of the three regular meals (breakfast dinner and supper).

The above outline should carry the average ulcer patient to about the end of the first week. In certain cases it may require 10 days to 2 weeks to advance the diet this far. Nevertheless for the sake of convenience the diet up to this point may be designated as *first week ulcer management*.

At the beginning of the *second week* (longer in some cases), an effort is made to develop three small meals breakfast dinner and supper. To breakfast and supper may be added 3 oz. of orange juice which has been diluted with an equal part of water and to which a teaspoonful of sugar has been added. Cream soups may be given at dinner. These additions are made in the first part of the second week and should be followed by adding more toast and butter 2 slices at each meal bland desserts such as custards simple puddings and gelatin and starchy vegetables that have been well cooked e.g. rice and potato with butter or margarine. This should carry the diet up to the end of the second week (longer in certain case) and for the sake of convenience terminates the *second week ulcer management*.

In the *third week*, breakfast, dinner, and supper feedings are still further increased in variety and quantity. Also, during this period the hourly milk and cream feedings are continued as in the beginning. To breakfast may be added jelly. At lunch, beans and peas may be given and for dessert baked apple without skin, applesauce, or tapioca pudding are allowed. The evening meal during the third week may likewise be increased by adding a cooked fruit or a simple dessert as at dinner. The above plan constitutes the *third week ulcer management*.

In the beginning of the *fourth week* (longer in some cases), the hourly feedings are discontinued and the patient is given instead three small meals (breakfast, dinner, and supper) and three supplemental feedings of equal part milk and cream, 6 to 8 oz., in the middle of the morning, in the middle of the afternoon, and at bedtime.

*Protein hydrolysates* as Amigen or Essamine may be administered parenterally entirely or orally when tolerated and at frequent intervals throughout the day. These will replace the blood proteins lost in a gastric hemorrhage and tend to stimulate more rapid healing of the ulcer.

**Protein Hydrolysate Therapy for Peptic Ulcer** (Hodges, Machella, and Miller) —The principle is to use high 50 % solutions of protein hydrolysate, delaying gastric emptying and thereby giving the solution time to completely neutralize gastric acidity until the next feeding. If weak solutions are used there will be a correspondingly more rapid emptying of the stomach with a subsequent rise in gastric acidity after a short while. The solution of about 50 % concentration, desirable in peptic ulcer is made up as follows:

To two quarts of boiling water (1892 cc) add one pound (454 Gm) of dextrin maltose (all carbohydrate) and one pound of Protolysate (Amigen, all protein). Stir well until dissolved. The solution must be clear and stored in the refrigerator. If it becomes cloudy, it is contaminated and must be discarded. It is an excellent culture medium.

The above recipe provides a 48 % solution, attains a total volume of about 2,350 cc, contains 454 Gm of protein, 454 Gm of carbohydrate and no fat. It is equivalent to 3,632 calories.

Each hour from 8 A.M. until 10 P.M. the patient is given a glass of about 150 cc, thus requiring about 2,100 cc for the day. If the solution looks clear the next day, the remaining portion may be used. It is best, however, to make the solution up fresh every day.

No other food, milk, or medication is given but vitamins, including vitamin K and iron are supplied in addition.

Two weeks is the usual course of therapy. On this regimen ulcers heal more rapidly than on the milk and cream diet, then too, the cholesterol consistently falls which is a desirable feature.

**Ulcerative Colitis and Regional Enteritis**—In ulcerative colitis and regional enteritis similar preparations in less concentrated form are used calculated to meet the caloric requirements of the patient. Potassium is supplied in the form of Valentine's meat extract in addition to the vitamins and iron as noted above since such patients are losing potassium in diarrhea. The meat extract is not given to the ulcer patients since the need is not so great and since the extract increases gastric acidity.

#### References

- Hodges H H Protein Hydrolysate Therapy for Peptic Ulcer. Report on 26 Cases Gastroenterology 2 4:6 193 April 1947
- Machella Thomas F Significance of Hyperalimentation in Treatment of Chronic Idiopathic Ulcerative Colitis Am J Med 7 191 197 Aug 1949
- Miller T Grier and Machella Thomas F Principles in the Management of Chronic Non Specific Ulcerative Colitis Tr A Am Physicians 61 203 1949
- Machella Thomas F and Miller T Grier Treatment of Idiopathic Ulcerative Colitis by Means of a Medical Ileostomy and an Orally Administered Protein Hydrolysate Dextrin Maltose Mixture Gastroenterology 10 28-40 Jan 1949
- Punge Thomas Marshall Personal communication

#### Routine Management of Patients With Gastric Hemorrhage

- 1 Keep patient quiet by ordering enough morphine to prevent thirst bromides and chloral hydrate given by rectum, may also be administered
- 2 Order gastric hemorrhage diet see above and below
- 3 Type patient's blood obtain donors match blood
- 4 Give no blood transfusions until after first week, unless indicated by air hunger imperceptible pulse or markedly lowered blood pressure
- 5 Determine Hb RBC and WBC every 2 days more often if hemorrhage persists
- 6 Determine coagulation and bleeding time every 2 days and before and after transfusion
- 7 Make no efforts at moving bowels for 3 days on 4th night order warm oil enema (3 oz to be retained), to be followed by small soap suds enema on the next day if necessary continue oil enema every night if needed
- 8 Test stools daily for occult blood until reactions are negative
- 9 Observe following measures
  - Avoid giving ice food or medicaments by mouth, except as indicated
  - Avoid sudden increase in blood volume by parenteral administration of fluids except as noted in 4



In the *third week*, breakfast, dinner, and supper feedings are still further increased in variety and quantity. Also, during this period the hourly milk and cream feedings are continued as in the beginning. To breakfast may be added jelly. At lunch beans and peas may be given, and for dessert baked apple without skin, applesauce, or tapioca pudding are allowed. The evening meal during the third week may likewise be increased by adding a cooked fruit or a simple dessert as at dinner. The above plan constitutes the *third week ulcer management*.

In the beginning of the *fourth week* (longer in some cases), the hourly feedings are discontinued and the patient is given instead three small meals (breakfast, dinner, and supper) and three supplemental feedings of equal part milk and cream, 6 to 8 oz., in the middle of the morning, in the middle of the afternoon, and at bedtime.

*Protein hydrolysates* as Amigen or Essamine may be administered parenterally entirely or orally when tolerated and at frequent intervals throughout the day. These will replace the blood proteins lost in a gastric hemorrhage and tend to stimulate more rapid healing of the ulcer.

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To two quarts of boiling water (1 892 c c) add one pound (454 Gm) of dextrin maltose (all carbohydrate) and one pound of Protolysate (Amigen, all protein). Stir well until dissolved. The solution must be clear and stored in the refrigerator. If it becomes cloudy it is contaminated and must be discarded. It is an excellent culture medium.

The above recipe provides a 48 % solution attains a total volume of about 2,350 c c, contains 454 Gm of protein, 454 Gm of carbohydrate, and no fat, it is equivalent to 3 632 calories.

Each hour from 8 A M until 10 P M the patient is given a glass of about 150 c c, thus requiring about 2 100 c c for the day. If the solution looks clear the next day, the remaining portion may be used. It is best, however, to make the solution up fresh every day.

No other food, milk, or medication is given but vitamins including vitamin K and iron are supplied in addition.



Avoid raising of blood pressure by use of epinephrine, ergot or blood stimulants, except in case of severe shock when stimulants can be given with transfusions

Avoid lowering of patient's body temperature and increase of shock by use of ice bags

10 Keep patient warm

### Andresen Treatment of Massive Gastric Hemorrhage Due to Peptic Ulcer

#### LONG ISLAND COLLEGE HOSPITAL DIET FOR PATIENTS WITH GASTRIC HEMORRHAGE

		GRAMS	OUNCES	CALORIES
<i>Gelatin Solution</i>	Gelatin	30	1	128 0
	Lactose	90	3	360
	Juice of 1 orange			47 8
	Water	1 000	32	
<i>Gruel Mixture 1</i>	Cereal gruel (oatmeal, barley, or cornmeal)	500		338 5
	Milk	420	16	290 8
	Cream	120	4	458 9
	Lactose	90	3	360
<i>Gruel Mixture 2</i>	Cereal gruel (same)	350	12	227 5
	Milk	1,000	32	602
	Cream	120	4	458 9
	Lactose	120	4	480

#### SCHEDULE OF FEEDINGS

		OUNCES	TOTAL CALORIES
<i>1st and 2nd days</i>	Feed every 1½ hr	Gelatin solution 4	947 2
<i>3rd day</i>	Feed q 1½ hr	Gelatin solution 4	
		Gruel 1 5	1,365 4
<i>4th day</i>	Feed q 1½ hr	Gelatin solution 5	
		Gruel 1 5	1,499 3
<i>5th and 6th days</i>	Feed q 1½ hr	Gelatin solution 6	
		Gruel 2 6	3 600
<i>7th and 8th days</i>	Feed q 2 hr	Gelatin solution 6	
		Gruel 2 6	3 600
<i>9th day and thereafter</i>		Ulcer diet, LIC Hospital	
Add to gruel mixture at each feeding one of the following		3 oz cereal, 1 soft poached egg custard or Jello	

#### ULCER DIET LIC HOSPITAL

<i>Breakfast</i>	Milk 8 oz with cream if desired cereal 5 oz with milk or cream 1 egg, soft boiled or poached, fruit juice or stewed fruit (end of meal)
<i>Midmorning meal</i>	Milk 8 oz, cream 1 oz lactose 1 oz with cocoa if desired all ways with crackers, toast bread or cake
<i>Luncheon</i>	Milk 8 oz, baked or mashed potato or plain spaghetti 1 egg soft boiled or poached or cream cheese bread and butter 3 slices, pudding custard gelatin ice cream or stewed fruit
<i>Midafternoon meal</i>	Same as midmorning meal
<i>Supper</i>	Same as breakfast or luncheon

Bedtime meal	Same as midmorning meal, repeat q 2½ hr during night if awake
Moderation	Olive oil ½ oz t.i.d. ac, liquid petrolatum ½ oz at bedtime, water as desired.

This treatment was devised by Andresen in 1916

#### References

- Andresen A. F. R. The Treatment of Gastric Hemorrhage J. A. M. A. 89  
139 148<sup>o</sup> Oct 2<sup>o</sup> 1927  
Ladue John S. Treatment of Massive Hemorrhage Due to Peptic Ulcer, Arch  
Int Med 63 1017 10 9 June 1939

### Modified Andresen Diet

Andresen mixture as served in most hospitals

- 900 cc milk
- 100 cc cream
- 60 Gm. dextrose
- 30 Gm gelatin

Formula above makes 1 quart

### Meulengracht Diet for Bleeding Peptic Ulcer

The following treatment is undertaken

From the day after their admission to the hospital all the patients are given a full puree diet together with a mixture consisting of sodium bicarbonate and magnesium subcarbonate  $\bar{\text{a}}$  10 Gm and extract hyoscyamine 2 Gm one teaspoonful three times daily also 0.5 Gm ferri lactas t d s

The puree diet includes the following meals

- 6 A.M. Tea white bread and butter
- 9 A.M. Oatmeal with milk white bread and butter
- 1 P.M. Dinner
- 3 P.M. Cocoa
- 6 P.M. White bread and butter sliced meats cheese and tea

The dinner includes a variety of dishes e.g., meat balls timbale broiled chops omelettes fish balls potatoes vegetable purees vegetable soups cream of vegetables stewed apricots applesauce gruel and rice and tapioca puddings. The patients are allowed to have as much as they want. In a few cases in which large quantities of blood have been lost they have been given one or two blood transfusions soon after admission.

#### Reference

- Meulengracht E. Treatment of Hematemesis and Melena With Food Lancet  
2 1 0 1945

**Modified Meulengracht Diet**

Following is the diet as prepared in most hospitals

Five meals are given daily, for example

7 30 A M	White bread, butter, and tea
10 00 A M	White bread, butter, and oatmeal with milk
12 30 P M	Omelet, meat balls, fish balls with fish or vegetables are au gratin, broiled chop, vegetable soups and purées, mashed potato, stewed apricots, applesauce, rice or gruel, tapioca pudding
3 00 P M	Cocoa
6 00 P M	White bread, butter, cheese, sliced meats, and tea

This diet is suitable for a bleeding peptic ulcer

**Sippy Ulcer Diet**

Milk and 40 % Cream, aa B 90 c c at 7, 8, 9, 10, 11, 12, 1, 2, 3 4, 5, 6, 7, 8 (9 10?) if awake

B Sod bicarb	{ 06 Gm aa 06 Gm at 7 30, 9 30, 11 30 1 30 3 30, 5 30 7 30, 9 30
Bismuth subcarb	
1/2 Mag Oxide	{ 03 Gm at 8 30, 10 30, 12 30, 2 30 4 30, 6 30, 8 30 06 Gm
Sod bicarb	

Regulate antacids to control acidity and pain Aspirate contents each evening two hours after last powder and estimate the acidity At tempt to get neutralization of acidity

Reference Sippy, Bertram W J A M A 64 1625 2090 May and June, 1911

**Modified Sippy Diet****MORNING**

HOURLY DAY	6	7	8	9	10	11	12
1st	Milk 60 c c	Albumin Water 90 c c	Milk	Albumin Water	Milk	Albumin Water	Milk
2	Cream 30 c c		Cream		Cream		Cream
3		1 egg					
4			Cereal 90 c c or Gm				
5							Uréa 90 c c or Gm
6		Bacon 2 slices— crisp					
7					Broth 90 c c		

## AFTERNOON AND EVENING

DAY	1	2	3	4	5	6	7	8	9	10
1.	Milk	Albumin Water	Milk	Albumin Water	Milk	Albumin Water	Milk	Albumin Water	Milk	Albumin Water
2.		Cream		Cream		Cream		Cream		Cream
3.							Egg			
4.		Cereal								
5.	Egg (1)					Purée				
6.	Bacon						Bacon			
7.				Broth						Broth

The diets for the eighth to the fourteenth days are similar to the diet of the seventh day with the addition of half a slice of toast at any three feedings. Custard 90 c.c. or Gm. may be substituted for any milk or albumin water feedings.

The food substances enumerated are added each day to the first and succeeding days' diets at the special hours.

## Methods of Preparing Foods and Notes

**Albumin Water**—To the white of one egg add enough water to make 90 c.c. Flavor with orange or lemon juice or vanilla. Use no sugar (Serve as albumin water in the schedule.)

**Cereals**—Cereals should be soft and well cooked. Measure after cooking. Ralston's Farina Cream of Wheat or boiled rice may be used.

**Purée**—Cream of peas, corn, spinach or celery (strained or run through a colander). Use no salt.

**Bacon**—Two slices which should be desalted and fried crisp.

**Broth**—(Clear) chicken, oyster or clam stock.

**Water intake**—Water intake is not limited.

**Medicine**—Give  $\frac{1}{2}$  teaspoonful of R. Sod Bicarb 50 Gm. Mag. Oxide (powd.) 50 Gm. in  $\frac{1}{4}$  glass of water after every feeding.

**Management**—Give nourishment every hour from 6 A.M. to 9 P.M. and on succeeding days additional feedings are to be taken at the hours indicated. Do not discontinue a feeding that has been started on any previous day. The amount of any food used as indicated in the first feeding thereof should remain constant.

## Hurst's Ulcer Program With a Modified Sippy Diet

Put teeth into perfect condition. Treat and eliminate any infected focus in the nose and throat.

Keep the mouth absolutely clean. Scrape the tongue with a wooden spatula if it becomes furred.

If an acute appendicitis recurs, appendix should be removed

Keep patient warm in bed, but allow up for daily bath and daily bowel movement

1 One hundred fifty c c (5 oz) of milk (or preferably milk and cream in equal parts) are given every hour from 8 A M to 8 P M inclusive

2 Add to each feeding

Sodium citrate 0.6 Gm (gr  $\times$ )

Emulsio magnesiæ 80 c c (drams 11)

Magnesiæ oxid (ponde) 0.6 Gm (gr  $\times$ )

The sodium citrate combines with the lime in the milk and consequently prevents the formation of irritating clots by rennin of the gastric juice

The calcined or heavy magnesium oxide has the advantage over sodium bicarbonate in having four times the neutralizing power, and in not giving off  $\text{CO}_2$  and distending, in having a mild aperient action, and in producing a very much smaller secondary increase in gastric secretion, after the initial neutralization than sodium bicarbonate does. The latter is the most powerful stimulant of gastric secretion in existence

3 Immediately before alternate feedings beginning at 7.30 A M, 15 c c ( $\frac{1}{2}$  oz) of olive oil is taken

4 Immediately before the remaining feedings and at 9.30 and 10 P M give a powder containing

Calcium carbonate 0.6 Gm (gr  $\times$ )

Bismuth carbonate 2.0 Gm (gr  $\times \times \times$ )

5 At 6 A M give a powder containing

B Bismuth carbonate 15 Gm ( $\frac{1}{2}$  oz)

Water 150 to 300 c c (5 to 10 oz)

When shaken up but not suspended and taken by mouth with the patient lying on his right side in a position such that the powder is likely to come in contact with the ulcer there is a protective covering formed

6 If there is pylorospasm with resulting stasis at 11 P M the stomach is completely emptied by Senoran's evacuator. If not more than 60 c c (2 oz) of fluid are present on two consecutive nights this can be discontinued. If 125 c c or more are removed at 11 P M the stomach should be evacuated again at 1 A M

7 At 11 P M if there is evidence of stasis atropine sulfate 0.001 Gm ( $\frac{1}{100}$  gr) is injected subcutaneously. At the same time the alkaline powder should be given and if more than 60 c c of fluid were evacuated, the powder should be repeated every two hours through the night

8 If the patient is constipated the dose of magnesia should be increased and if the bowels do not move on two consecutive days an enema should be given. If diarrhea occurs some of the magnesia should be replaced by an *equivalent* amount of bismuth carbonate.

9 This strict treatment is carried out until for three weeks the patient has had no spontaneous pain no trace of tenderness has been present no occult blood has been found in the stools not more than 60 c.c. of fluid has been removed on any one evening and the x ray shows no evidence of active ulceration. Then the diet can be rapidly increased omitting however all irritants.

Reference Hurst Arthur F. Brit. M. J. p. 539 April 24 1908

### Mills Ulcer Diet

#### List No. 1

Used in acute cases

Breakfast —

- Oatmeal jelly (well cooked strained)
- Soft boiled eggs (cooked seven minutes)
- Hot water and cream ( $\frac{1}{2}$  cream)

10.30 A.M. —

- Glass of half milk and cream

Noon —

- Soft boiled eggs (cooked seven minutes)
- Toast soaked in hot water add cream
- Custard gelatin blancmange corn starch
- Hot water and cream ( $\frac{1}{2}$  cream)

4 P.M. —

- Glass of half milk and cream

Night —

- Same as at noon

#### List No. 2

Used in subacute cases

Breakfast —

- Thoroughly cooked and strained Cream of Wheat or Farina with cream and little sugar
- One soft boiled egg (cooked seven minutes)
- One cup hot water and cream ( $\frac{1}{2}$  cream)

10.30 A.M. —

- Crumbled crackers in warm milk or crumbled stale bread (soft part only) in warm milk or glasses of  $\frac{1}{2}$  lactose or Fermilla or milk and  $\frac{1}{2}$  cream

Noon —

- Milk toast
- Thin dry toast and butter



If an acute appendicitis recurs, appendix should be removed

Keep patient warm in bed, but allow up for daily bath and daily bowel movement

1 One hundred fifty cc (5 oz) of milk (or preferably milk and cream in equal parts) are given every hour from 8 A M to 8 P M inclusive

2 Add to each feeding

Sodium citrate 0.6 Gm (gr v)

Emulsio magnesiæ 80 cc (drams ii)

Magnes. oxid. (ponde) 0.6 Gm (gr v)

The sodium citrate combines with the lime in the milk and consequently prevents the formation of irritating clots by rennin of the gastric juice

The calcined or heavy magnesium oxide has the advantage over sodium bicarbonate in having four times the neutralizing power, and in not giving off CO<sub>2</sub> and distending in having a mild aperient action and in producing a very much smaller secondary increase in gastric secretion, after the initial neutralization, than sodium bicarbonate does. The latter is the most powerful stimulant of gastric secretion in existence

3 Immediately before alternate feedings beginning at 7.30 A M, 15 cc (½ oz) of olive oil is taken

4 Immediately before the remaining feedings and at 9, 9.30 and 10 P M give a powder containing

Calcium carbonate 0.6 Gm (gr v)

Bismuth carbonate 2.0 Gm (gr xxv)

5 At 6 A M give a powder containing

R Bismuth carbonate 15 Gm (½ oz)

Water 150 to 300 cc (5 to 10 oz)

When shaken up, but not suspended and taken by mouth with the patient lying on his right side, in a position such that the powder is likely to come in contact with the ulcer, there is a protective covering formed

6 If there is pylorospasm with resulting stasis at 11 P M, the stomach is completely emptied by Senoian's evacuator. If not more than 60 cc (2 oz) of fluid are present on two consecutive nights, this can be discontinued. If 125 cc or more are removed at 11 P M, the stomach should be evacuated again at 1 A M

7 At 11 P M, if there is evidence of stasis atropine sulfate 0.001 Gm (⅓ gr) is injected subcutaneously. At the same time the alkaline powder should be given and if more than 60 cc of fluid were evacuated, the powder should be repeated every two hours through the night

noon —

Eggs poached soft boiled (coddled seven minutes) omelet  
 Cottage cheese with cream  
 Creamed spinach, asparagus tender string beans peas well mashed squash,  
 stewed cream white onions, stewed creamed carrots  
 Graham or whole wheat bread and butter  
 Stewed apples stewed prunes baked apples  
 Custards gelatins, cornstarches, tapioca, Junket, blancmange  
 Soft ice cream with bread and butter  
 One cup hot water and cream ( $\frac{1}{2}$  cream) or  
 Glass of cool water

night —

Tender roast beef, roast lamb roast chicken (without dressing) or stewed  
 creamed chicken  
 Broiled steak, broiled lamb chops (lean portion only)  
 Stewed sweetbreads or calves brains  
 Baked fresh fish. (Crappie Bass or Whitefish)  
 Potatoes well mashed or baked or rice well cooked  
 Other vegetables same as at lunch  
 Graham or whole wheat bread and butter  
 Desserts same as at lunch  
 One cup hot water and cream ( $\frac{1}{2}$  cream)

Reference Mills R Walter Barnes Hospital St Louis Mo Personal Communication

### Diets for Intestinal Disorders Basic Bland Nonresidue Forming Diet

*First Day* — Every three hours

Milk	60 cc
Uneda biscuit	4
Sugar	qs

Give crackers to patient to break into bowl of milk add sugar to  
 taste and feed every three hours

*Second Day* —

Increase the above diet in frequency and amount so as to total twice  
 the amount of the first day

If necessary keep up for two weeks to relieve gastric symptoms

### Low Residue Forming or Nonlaxative Foods for Diarrheal Cases

Clear Meat Soups	Eggs
Mashed or Baked Potatoes	Macaroni
Rice	Spaghetti
Cream of Wheat	Cheese
Cornmeal Mush	Lean Meats
White Breads	Bananas
Milk (preferably boiled)	

Oatmeal jelly, or oatmeal (most thoroughly cooked strained, and allowed to cool)

Cup of hot water with cream ( $\frac{1}{2}$  cream)

4 P M —

Same as at 10 30 A M

Night —

Two soft boiled eggs (coddled seven minutes)

Thin dry toast and butter

Custards, gelatin cornstarches, Junket, blancmange

Cup hot water and cream ( $\frac{1}{2}$  cream)

10 P M —

Same as at 10 30 A M

### LIST No 3

Used during convalescing stages when free from pain

*Breakfast —*

Thoroughly cooked Cream of Wheat, Farina or

Corn Flakes, with cream and a little sugar

Eggs soft boiled (coddled seven minutes)

Stewed prunes—skins not to be eaten

Milk toast (made from stale white bread) and served with unsalted butter

One cup hot water and cream ( $\frac{1}{2}$  cream)

*Noon —*

Eggs soft boiled (coddled seven minutes)

Tender roast breast of turkey, chicken or quail (without dressing)

Scraped beef (mixed with the white of an egg and broiled)

Fresh fish baked white meat only

(Crappie, Bass, Whitefish)

Rice well cooked

Thin dry toast and unsalted butter

Milk toast and unsalted butter

Stewed prunes—skins not to be eaten

Custards, gelatins, cornstarches tapioca, Junket, blancmange all made with but little sugar

One cup hot water and cream ( $\frac{1}{2}$  cream)

*Night —*

Same as at noon

Use butter and cream freely All foods must be very well cooked

### LIST No 4

Used during interim between ulcer attacks and on which patient is kept indefinitely

*Breakfast —*

Stewed apples (not tart), stewed prunes

Shredded Wheat Biscuits well cooked Cream of Wheat Farina or Quaker

Oats with cream and sugar

Eggs poached, soft boiled (coddled seven minutes)

Toasted graham or whole wheat bread and butter

One cup hot water and cream ( $\frac{1}{2}$  cream)

	Water	300 c.c.
	Salt q.s.	
	Cook, strain, and add sugar to taste	
12 noon	Beef lean fine chopped cooked with butter	100 Gm.
	White potato	200 Gm.
	Milk	100 c.c.
	Butter	10 Gm.
5 P.M.	Same as 6:30 A.M.	
8 P.M.	Same as at 7:30 with Egg 1 beaten into oatmeal gruel.	

### Schmidt's Pancreatic Function Diets

Schmidt No 1 Diet (Pratt)—Protein 102 Gm, Fat 132 Gm, Carbohydrates 180 Gm Calories 2324

Schmidt No 2 Diet (Pratt)—Protein 118 Gm, Fat 111 Gm, Carbohydrates 190 Gm Calories 2234

Carmine 0.5 Gm capsules—two are given with the first meal of the diet. Charcoal is given in milk on the 4th day to mark the end of a 3 day test regime on the diet. The time between ingestion of the pigments and the appearance in the stool indicates the rate of passage of food through the G.I. canal and the motility.

The total fecal mass is weighed, dried, reweighed and divided into three equal parts and extracted with ether in a Soxhlet apparatus.

A weighed portion of the ether extract is dissolved in acid alcohol and titrated with standard alcoholic KOH with phenolphthalein as indicator for fatty acid. This subtracted from the total gives the neutral fat.

Soaps may be determined on an aliquot of the ether extract from the Soxhlet apparatus. Boil with HCl alcohol dry, and re-extract with ether. The fatty acids are split off from the soaps by HCl and after adding alcohol the amount of fatty acid obtained from soaps can be estimated by titration and the results changed by calculation into the equivalent amounts of soaps.

Results on the Schmidt fat diet—10 to 25 Gm of fat are excreted daily. Of dry feces 25 to 10 % are of neutral fat, 1 to 10 % are of fatty acid and 0.5 to 10 % are of soaps. Total fat is 17.5 % of dry feces.

In obstructive jaundice the total fat rises to 50 % of which soaps make up 20 % fatty acids 20 % and neutral fat 5 %.

In pancreatic insufficiency the total fat rises to 45 % of which neutral fat makes up 30 % fatty acids 5 % and soaps 10 %.

## Schmidt Strasburger Diet

## DIET No 1

Same as Diet No 2 except that meats and potatoes are omitted

## DIET No 2

6 30 A M	Milk 375 c c				
	Zwieback (2 slices)	66 Gm			
9 30 A M	Bouillon, 375 c c				
	$\frac{1}{2}$ egg				
11 00 A M	Milk 375 c c				
	1 egg				
12 00 NOON	Oatmeal gruel, prepared from	Oatmeal	40 Gm	} 500 c c	
		Milk	166 c c		
		Sugar	10 Gm		
		Egg	$\frac{1}{2}$		
	Hamburger steak (well done)				
	prepared from	Raw beef	100 Gm	} 100 Gm	
		Butter	12 Gm		
	Mashed potatoes prepared from	Potatoes	190 Gm	} 250 c c	
		Milk	60 c c		
		Butter	8 Gm		
3 30 P M	Milk, 375 c.c.				
	Egg, 1				
	Zwieback, 1 slice (33 Gm )				
6 30 P M	Oatmeal gruel, 500 c c (prepared as above)				

Total make up

Milk	1 5 Liters	} {	Protein	126 25 Gm
Zwieback	100 Gm		Fat	83 4 Gm
Butter	20 Gm		Carbo	218 5 Gm
Sugar	20 Gm			
Steak (raw wt )	125 Gm			
Potatoes (raw wt )	190 Gm			
Calories				2,183 8

## References

- Strasburger, J Deutsch Arch f klin Med 61 584, 1868  
 Schmidt, Ad, and Strasburger J Die Faeces des Menschen mit klinischen Untersuchungsmethoden Berlin, 1903

## Schmidt s Gastro intestinal Function Test Diet

7 A.M	Milk	500 c c
	Roll, 1	
	Butter	10 Gm
	Egg 1 soft boiled	
	Tea	250 c c
8 A M	Oatmeal	40 Gm
	Butter	10 Gm
	Milk	200 c c

Oatmeal	Green corn
Pettigohn	Beets green
Bran	Squash
Bran bread	Swiss chard
Bran muffins	
Whole wheat bread	
Molasses	
Honey	

Any of the following desserts may be eaten

Simpl apple or fig or prune puddings  
Cautards  
Sherbets  
Fruit  
Gelatin  
Fruit whips  
Raisin pudding  
Gingerbread

Mix a small quantity of agar agar Kellogg's bran or Plantago ovato with the breakfast cereal Agar agar mixed with jam or marmalade is also beneficial

It is well to use milk sugar in preference to cane sugar in the preparation of foods Fruit should be stewed with honey molasses, or milk sugar

Do not eat concentrated low residue foods Cheese pineapple rice, rice puddings new bread tapioca pastry salt or smoked meat or fish dried beans or peas dry vegetables berries nor an excessive amount of macaroni spaghetti barley milk or eggs

The addition of agar agar or psyllium seeds or flakes or pellets of sterculia gums from the Plantago ovato plants provide natural laxative and hydrophilic lubricated bulk

The salines are galenicals that disturb the physiology of the bowel and produce the cathartic habit which requires daily medication and results in gas Such drugs should be avoided

The enema habit is almost as bad and should be stopped and the natural laxatives fruits and vegetables substituted

### Diet in Diabetes Mellitus

Diet is the most important part in the management and treatment of patients with diabetes mellitus It must be adjusted to the individual patient and the physical effort that will be required of him He must be given enough calories to do his job and if necessary insulin must be added to provide efficient utilization of glucose by the patient's body The patient's metabolism must be brought back as near to normal

In diarrhea, the total fat may reach 48 % with neutral fat 13 %, free fatty acids 18 % and soaps 17 %

Fat absorption is deficient if soaps and fatty acids together total over 75 % of the total fat or over 15 % of the total dried feces. There is deficient fat splitting if neutral fat is over 55 % of total fat or 11 % of total dried feces

### Dietary Management of Constipation

Upon arising, drink one or two glasses of water, preferably warm. Drink plenty of water through the day, no less than 15 glasses, preferably between meals. On retiring, several glasses of water should be taken along with some fruit. Salads, with plenty of olive oil as a dressing, make a valuable bulk. Eat either fruit or a fresh vegetable, or both, with each meal.

An effort should be made to move the bowels daily at a definite hour, whether there is a desire to do so or not. The best time for this is immediately after breakfast. This procedure should be persisted in and the effort continued for as long as ten minutes at a time, even if the bowels do not move.

The following exercises should be taken. Follow them with a cool sponge or plunge.

#### Exercises —

1 Stand erect with the arms extended and the fingers in contact above the head. Stoop forward and try to touch the floor with the finger tips, moving slowly and return again to the original position.

2 Lie at full length on the bed or floor upon the back, with the hands under the hips, and bring each leg alternately and slowly at right angles to the body.

3 Standing position, extend the arms sideways with the elbows straight and raise slowly until the hands meet above the head. Rise on tiptoe at the same time. Stretch the arms as much as possible in doing this. Inhale deeply as the arms go up, and exhale slowly as the arms go down.

Select diet from the following high residue containing foods —

Oranges	Lettuce	Fresh fish
Grapefruit	Cucumbers	Fresh meat
Apples	Cabbage	Eggs (not fried)
Pears	Celery	Buttermilk
Peaches	Asparagus	Salads with olive oil
Stewed prunes	Spinach	Raisins
Stewed apricots	String Beans	Figs
Applesauce	Onions	
Stewed rhubarb	Brussels sprouts	

and teach the patient to weigh his foods. Total glucose, fat and protein allowance may be divided into three equal parts for the three meals. They may be planned by first dividing the total glucose value for the day into three parts for the three meals. Calculate the carbohydrates for each meal and then divide the protein and finally the fats. If an evening snack or glass of milk is taken the food values must be subtracted from other meals. At least 300 Gm of low percentage carbohydrate food especially vegetables and fruits should be included in order to furnish the necessary vitamins minerals and bulk.

Proteins should be from different sources and types as beef ham and pork fish fowl dairy products and cheese. Food values and household measurements are given in the same diet tables. The patient should not vary more than 10 % above the calculated diet or below it for the day. Tea and coffee without cream and sugar clear broth, salt pepper paprika and vinegar have no food value. Saccharin or crystalline may be used to sweeten beverages. Likewise whisky in water and dry wine may be added to the diet. D Zerta water packed fruits or those fruits canned in their own juice and without added sugar syrup are supplied.

THE BASIC DIABETIC DIET

	HOUSEHOLD MEASUREMENTS	GRAMS	CARBOHY- DRATE	PROTEIN	FAT
<i>Breakfast</i>					
Orange	$\frac{1}{2}$ cup	150	15	-	-
Egg	1	1	-	6	6
Cream 10 %	4 Tbsp	90	3	3	19
Bacon	1 slice	15	-	2.5	7.5
Butter	2 t p	10	-	-	8
			18	11	33.5
<i>Dinner</i>					
5 % vegetables	$1\frac{1}{2}$ cup	300	10	5	-
Orange	1 small	75	7.5	-	-
Cream 10 %	4 Tbsp	90	3	3	18
Meat (4 x 2 x 1)		60	-	16	10
Butter	tsp	10	-	-	8.5
			20.5	4	36.5
<i>Supper</i>					
5 % vegetables	$1\frac{1}{2}$ cup	300	10	5	-
Orange	1 small	75	7.5	-	-
Egg	1	1	-	6	6
Cream 20	4 Tbsp	90	3	3	18
Bacon	1 slice	15	-	2.5	7.5
Butter	2 t p	10	-	-	8.5
			0.5	16.5	40.0
Total for Day			59	5	116
Total Calories			36	4	9
				68	1041 or 1488



as possible Dietary treatment and science have come a long way since the early starvation, low carbohydrate diets of Allen the high fat diets of Newburgh, the high carbohydrate diets of the early insulin days Present day diabetic diets are planned on the basis of the more rational two thirds the normal content of carbohydrate, low fat, and higher proportion of protein than were previously employed

The protein requirement is now calculated at first, carbohydrate allowance next, and the fat added to obtain necessary caloric levels Adequate diet is one that contains enough of the necessary food elements to maintain a sense of well being, normal efficiency, endurance, and weight This usually cannot be accomplished with diet only in the moderately severe or severe diabetic patient without insulin to prevent the loss of sugar in the urine and elevation of the blood sugar Insulin must be added to the point where the necessary diet is tolerated by the patient the urine remains sugar free and the blood sugar normal In the presence of infections or acidosis, undernutrition or obesity special modifications in the diet of the diabetic and insulin are indicated In general, the diet is peculiar to each individual It depends upon his weight height age, and general condition

The principles in diabetic diets thereby consist in the establishment of basal requirements for the individual Published charts may be consulted Protein requirements for adults are 1 Gm up to 2 Gm per kilogram of body weight In children it is at least 1 to 3 Gm per kilogram Carbohydrates sufficient to supply no less than 120 Gm and for patients taking insulin not less than 150 Gm of glucose These are minimum values and may be modified upward according to the patient's tolerance The total caloric value of the diet depends upon the patient's occupation For sedentary individuals 20 to 40 calories per kilogram is adequate for adults, while in children it is about 40 to 70 calories per kilogram If he becomes active 50 % more calories are needed It may be added to bring up the total caloric levels It is desirable to keep it in the proportion of  $1\frac{1}{2}$  to 3 to 1 Calories must be sufficient to maintain the patient's health slightly below the average weight for his age and height In sedentary occupation, 30 calories per kilogram moderately heavy activity, 35 calories per kilogram, heavy physical work, 40 calories per kilogram The Eli Lilly Company will supply upon request a slide rule for making all these calculations and a book of diets and a valuable monograph

In *planning the menu*, one must begin by having a dietitian prepare a diet according to weight, upon prescription from the physician Established fixed diets of all practical proportions are available in book form but it is desirable for the physician to work out the diet with the patient

Canned grapefruit juice  
 Oranges and orange juice  
 Fresh peaches  
 Pineapple or pineapple juice  
 Fresh plums  
 Fresh raspberries  
 Fresh blueberries  
 Apples  
 Grapes  
 Fresh pears  
 Canned black cherries

*Group V (¼ cup)*

Fresh cherries  
 Fresh figs  
 Grape juice  
 Bananas  
 Stewed prunes  
 Canned prune juice

*Group III (½ cup)*

Beets  
 Brussels sprouts  
 Fresh carrots  
 Onions  
 Fresh young peas  
 Canned green peas  
 Rutabagas

*Group IV (¼ cup)*

Canned green lima beans  
 Fresh young corn  
 Fresh or frozen green peas  
 Parsnips

NOTE All fruit must be fresh or canned without sugar and vegetables should be seasoned with part of the butter allowed on the diet

Diabetic Diets

1000 Calorie Diabetic Diet

CHO—90 Prot—68 Fat—4.

*Breakfast*

Fruit — One serving from the preceding list  
 Egg — One cooked any way except fried or cooked with grease  
 Toast — One half slice  
 Butter — One level teaspoon  
 Cakes with saccharin or crystallose

*Dinner & Supper*

Lean meat — One medium serving without fat or gravy and not fried or  
 Two eggs or  
 a serving of cottage cheese and leave off butter  
 Vegetable — Anyone from those listed in previous section  
 Bread — One half slice  
 Butter or margarine — One level teaspoon  
 Skim milk or buttermilk — One glass  
 Fruit — One serving from the preceding list

1200 Calorie Diabetic Diet

CHO—112 Prot—72 Fat—42

*Breakfast*

Fruit — One serving from the list given above  
 Egg — One cooked any way except fried or cooked with grease  
 Toast — One slice

by the Health Food Centers who cater to the patient with diabetes mellitus (Cellu Products, etc.)

*Vegetables* that are cooked should be weighed after cooking. Excess fats should be removed from the meat. Bacon should be fried crisp and weighed. Margarine should be substituted for butter. In general, plain foods are preferable to compounded or processed foods. The usual vegetable groups and caloric food values and menus covering the usual distribution of carbohydrate, protein, and fat will be given in the pages of the sections that follow.

Regular insulin should be taken 20 minutes before meals, and meals should follow within 20 minutes. With protamine zinc insulin or globin insulin or NPH 50 (Lilly), this is not so necessary. It is well to determine roughly the amount of glucose in the urine one half hour before each meal and judge the insulin dosage accordingly. Five units for a green reaction, 10 units for a yellow test, 15 units for an orange reaction, and 20 units for ordinary insulin are given 20 minutes before the meal if a brick red precipitate develops in the qualitative Benedict's solution or Clinutest.

#### List of Servings of Fruits and Vegetables for Diabetic Diets

FRUITS	VEGETABLES
<i>Group I (1½ cups)</i>	<i>Group I (1½ cups)</i>
Rhubarb	Asparagus
	Canned green beans
<i>Group II (¾ cup)</i>	Beet greens
Canned blackberries	Broccoli
Cantaloup	Cabbage
Watermelon	Cauliflower
Honeydew melon	Celery
Canned peaches	Cucumbers
Canned plums	Lettuce
Fresh strawberries	Mustard greens
	Radishes
<i>Group III (½ cup)</i>	Sauerkraut
Stewed apples & applesauce	Spinach
Canned apricots	Summer squash
Fresh blackberries	Tomatoes & tomato juice
Canned red & white cherries	Turnip greens
Fresh cranberries	
Grapefruit	<i>Group II (¾ cup)</i>
Fresh grapefruit juice	Fresh green beans
Lemon and lime juice	Canned carrots
Canned pears	Eggplant
Tangerines	Okra
	Green & red peppers
<i>Group IV (⅓ cup)</i>	Fresh & canned pumpkin
Fresh apple juice	Winter squash
Fresh apricots	Fresh turnips
Canned figs	

Butter	-- -- --	Two level tea spoons
Cream for coffee	-- -- --	Two tablespoons
Coffee with saccharin or crystalline		
<i>Dinner</i>		
Lean meat	-	One piece 4 x 2½ x ½ inches, without fat or gravy or One egg plus 2 rounding tablespoons cottage cheese
Potatoes corn noodles rice macaroni spaghetti grits blackeyed peas or dried beans	-- -- --	½ cup
Fresh or canned vegetable or salad	-	One serving as on the list on p 434
Bread	-- -- --	One slice
Butter margarine or mayonnaise	-	Two level teaspoons
Fruit	-- --	One cup of any listed on p 434
Milk	-	One cup

*Supper*

Same meal as at noon except that you may increase bread to 1½ slices and butter to 2 level teaspoons

*1,800 Calorie Diabetic Diet*

CHO—176 Prot—80 Fat—87

<i>Breakfast</i>		
Fruit	-	One serving as listed.
Cereal	-	½ cup cooked or ¾ cup dry
Eggs	-	Two, cooked in any way desired but if fried, use part of the butter allowed
Toast or bread	-	One slice
Butter or margarine	-	Two teaspoons
Cream for cereal or coffee	-	¾ cup
Coffee with saccharin or crystalline		
<i>Dinner and Supper</i>		
Lean meat	-	One piece 4 x 2½ x ½ inches without fat or gravy or One egg plus 2 tablespoons cottage cheese or One and one half slices yellow cheese and leave off 1 teaspoon butter
Potatoes corn rice grits spaghetti macaroni, blackeyed peas or dried beans or peas	-- -- --	½ cup
Fresh or canned vegetable or salad	-	One serving as on list
Bread	-	1½ slices
Butter margarine or mayonnaise	-	Two teaspoons
Milk	-	One cup
Fruit	-	One serving as on list

*1,000 Calorie Diabetic Diet*

CHO—193 Prot—80 Fat—100

<i>Breakfast</i>		
Fruit	-	One serving as listed
Cereal	-	½ cup of cooked or ¾ cup dry
Cream for cereal and coffee	-	¾ cup

Butter or margarine .....	One level teaspoon
Cream .....	Two tablespoons
Coffee with saccharin or crystallose	

*Dinner*

Lean meat .....	One serving of any kind without fat or gravy, or Two eggs, or a serving of cottage cheese with cream, or a thin slice of yellow cheese and leave off 1 tea spoon of butter
Vegetable .....	One serving from the list on p 434
Bread .....	One slice
Butter .....	One level teaspoon
Skim milk or buttermilk .....	One glass
Fruit .....	One serving from the list on p 434

*Supper*

Take the same meal as at noon but you may increase your butter to 2 level teaspoons

*1400 Calorie Diabetic Diet*

CHO—132 Prot—74 Fat—65

*Breakfast*

Fruit .....	One serving as listed
Egg .....	One, may be prepared in butter allowed on diet
Bread .....	One average slice
Butter .....	One level teaspoon
Cream .....	Two tablespoons
Coffee with saccharin or crystallose	

*Dinner*

Lean meat .....	One piece $4 \times 2\frac{1}{2} \times \frac{1}{2}$ inches, without fat or gravy, or One egg plus two rounding tablespoons of cottage cheese
Fresh or canned vegetable or salad..	One of any of the servings listed
Bread .....	One average slice
Butter, margarine, or mayonnaise..	Two level teaspoons
Fruit .....	One of any of the servings listed
Milk .....	One cup

*Supper*

Same as noon meal except one may add  $\frac{1}{4}$  cup of potatoes, rice, noodles, grits, spaghetti, blackeyed peas, dried beans, corn, or macaroni

*1,500 Calorie Diabetic Diet*

CHO—158 Prot—72 Fat—77

*Breakfast*

Fruit .....	One of any on the list
Egg .....	One, may be cooked in butter allowed on the diet
Bread .....	One average slice, toasted or plain

*\* 400 Calorie Diabetic Diet*  
CHO—997 Prot—91 Fat—126

## Breakfast

Fruit	- -	One serving as listed
Cereal	- -	$\frac{1}{3}$ cup cooked or $1\frac{1}{3}$ cups dry
Cream	- - - -	$\frac{1}{2}$ cup
Eggs	- -	Two
Bacon	- -	Two crisp strips, five inches long
Bread or toast	- -	Two slices
Butter or margarine	- - -	Five level teaspoons
C See with accharin or crystalline		

## Dinner

Lean meat	- - - - -	One piece $4 \times 3 \times \frac{1}{2}$ inch without fat or gravy or Two eggs or One medium slice of yellow cheese and omit 2 tsp butter or one large serving cottage cheese with $\frac{1}{4}$ cup cream
Potatoes spaghetti, macaroni	noodles rice blackeyed peas, dried beans or grits	$\frac{1}{3}$ cup listed on p 434
Green or yellow vegetable or salad		Two servings as listed or a double serving of one vegetable
Bread		$1\frac{1}{2}$ slices (or may take two slices of bread and only one serving of vegetable if preferred)
Butter margarine or mayonnaise		3 level teaspoons
Milk		1 cup or an ordinary glass
Fruit		One of the servings as listed on p 434

## Supper

Prescribe the same meal as at noon except for an increase in bread to 2 slices (or  $2\frac{1}{2}$  slices if only one serving of vegetable is taken from the sheet) and the serving of fruit may be increased to  $1\frac{1}{2}$  times the amount listed on the fruit and vegetable sheet

*3 000 Calorie Diabetic Diet*  
CHO—24° Prot—116 Fat—136

## Breakfast

Fruit	-	Any one of the servings as listed on the Fruit list
Cereal	-	$\frac{1}{3}$ cup cooked $1\frac{1}{3}$ cups dry
Eggs	- -	Three cooked any way desired but if fried use part of butter allowed on the diet
Bacon		Two crisp strips about 5 inches long
Bread or toast		Three slices white or whole wheat
Butter or margarine		Two tablespoons
Milk		One cup

## Dinner and Supper

Meat	- -	One large serving of lean meat without fat or gravy or Three eggs or A slice of cheese about $3 \times 2\frac{1}{2} \times \frac{1}{2}$ inches or A large serving cottage cheese with cream
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Eggs	Two, cooked any way desired, but if fried, use part of butter allowed on the diet
Toast or bread	One and one half slices
Butter or margarine	Three teaspoons
Coffee with saccharin or crystallose	

*Dinner*

Lean meat	One piece $4 \times 2\frac{1}{2} \times \frac{3}{4}$ inches, without fat or gravy, or $\frac{1}{3}$ cup cottage cheese plus 2 table spoons cream, or Two eggs, or $1\frac{1}{2}$ slices yellow cheese, $4 \times 4 \times \frac{1}{2}$ inches and leave off 1 teaspoon butter
Potatoes, rice, macaroni, noodles, grits, dried beans blackeyed peas, or spaghetti	$\frac{1}{4}$ cup
Raw or cooked vegetable or salad	One serving as listed on p 434
Bread	$1\frac{1}{2}$ slices
Butter, margarine, or mayonnaise	Three level teaspoons
Milk	One cup
Fruit	One serving as listed on p 434

*Supper*

Take the same meal as at noon but increase the bread to two slices

*2,200 Calorie Diabetic Diet*

CHO—217 Prot—92 Fat—109

*Breakfast*

Fruit	One serving as listed
Cereal	$\frac{1}{4}$ cup cooked or $\frac{3}{4}$ cup dry
Cream	$\frac{1}{2}$ cup
Eggs	Two, may be cooked in part of butter allowed
Bacon	Two crisp strips five inches long
Bread	One and one half slices, toasted or plain
Butter or margarine	One level tablespoonful
Coffee with saccharin or crystallose	

*Dinner*

Lean meat	One medium serving without fat or gravy or A medium serving of cottage cheese with cream, or Two eggs, or A slice of yellow cheese and omit 2 tsp butter
Potatoes, rice, grits, noodles, macaroni, dried beans, blackeyed peas spaghetti, or corn	$\frac{1}{4}$ cup
Canned or fresh vegetable or salad	Two servings as listed or a double serving of one vegetable
Bread	One and one half slices
Butter or margarine	Three level teaspoons or one level tablespoon
Milk	One cup or one ordinary glass
Fruit	One serving as listed

*Supper*

Same meal as at noon, choosing different vegetables, etc

body weight per day. Proteins of good biological value contain methionine and choline. These may be found in good quantity in skimmed milk, cottage or Swiss cheese and lean meat. Protein hydrolysate concentrates choline and methionine themselves which are now quite reasonably priced might be added. Carbohydrates should furnish abundant calories which are desired at the same time to spare protein and fats and store glycogen. The cirrhosis diet should contain 300 or 400 Gm of carbohydrates which is equivalent to and spares 200 Gm of proteins. The fat soluble and water soluble vitamins are necessary in normal metabolic processes and may help to free the liver of fatty deposits and stimulate liver cellular regeneration.

Crude liver extract contains valuable elements which include amino acids of the special type or parts of vitamin B complex, methionine, choline, and inositol which are essential agents in fat mobilization and metabolism. These hypotrophic substances are the active principles in the hypoprotein diet.

The hypotrophic agents alone as choline and methionine react specifically in the mobilization and removal of fatty deposits from the liver. However, sometimes the improvement is very slow and the patient should be reassured and the situation explained to the nearest of kin. The regime or program must be carried out meticulously and carefully with attention to details.

In general, present day diabetic mellitus therapy, as well as in cirrhosis, fatty foods are to be avoided, especially lard, butter, cream, rich milk, fats, pork, sausages, gravies, fried and greasy foods. Vegetable fats too should be cut to a minimum: margarine, hydrogenated cotton seed oil, shortenings, cocoa, nuts, olive oil, olives, avocados, coconut oil, as well as peanut or corn oil.

Restrictions on all highly seasoned, processed, prepared or canned foods are justifiable because of the high fat content of such foods.

Ice cream, pastries, pies, cakes, cookies, doughnuts, chocolate candy, peanut butter, strong coffee, tea and alcoholic beverages are to be avoided.

Generally speaking, poultry and fish should have the skin removed and be steamed, baked or boiled. Lean meats, especially beef, cheese, cottage cheese, skimmed milk or powdered skimmed milk should be used freely. Green vegetables, either fresh or frozen, should not be cooked with pork fat or olive oil but with bouillon cubes unless salt is restricted.

Starchy vegetables, including potatoes, sweet or Irish, boiled or baked with the jackets on, Spanish rice, macaroni, spaghetti, barley, cooked whole grain mixed cereals are unrestricted. Citrus fruits such



Potatoes, rice, corn, macaroni, noodles, grits, dried beans or blackeyed peas	One cup
Vegetables or salads	Two servings as listed on the Fruit and Vegetable List
Bread	Three slices
Butter	Three level teaspoons
Milk	One cup
Fruit	Any one of the servings given on the Fruit List

### Dietary Management in Liver Dysfunction

#### TREATMENT OF FATTY CIRRHOSIS OF THE LIVER

The nutritional basis of fatty infiltration of the liver related to liver dysfunction was first realized by Gyorgy and his associates, and by Patek and Post. The Toronto group, Allan, Fischer, Hershey, Huntsman, and Best, working with experimental diabetic and other fatty cirrhosis of dogs, opened new vistas in investigation of fat metabolism and therapy.

Patek and Post, Broun, Herrmann and Rockwell, Morrison, Chester Jones and his group in Boston have proved that fatty cirrhosis of nutritional failure may be due to alcoholism, diabetes, anemia and choline and methionine deficiency. This process is usually reversible even when the patient has progressed to the advent of ascites.

The ascitic state and fatty cirrhosis of the liver may be completely cured by a diet rich in vitamins, proteins and carbohydrates, and low in fat whether or not supplemented by lipotropic agents for mobilization of fat in the liver. Fatty infiltration in the liver, if it persists, stimulates fibrous tissue development and gradually changes into the irreversible hobnail Laennec cirrhosis of the liver.

Steps should be taken to prevent development of fibrous tissue in the liver by removing the causes of the active fatty cirrhosis. The management consists in the elimination of the alcoholic intake which has led to a nutritional imbalance, anorexia, and a diet low in choline and methionine. Prevent further exposure to the toxic agent itself that induced fatty degeneration. All animal fats should be removed and the intake of all fats should be sharply reduced. The blood protein level should be brought back to normal and if necessary, supplementary plasma or amino acids should be administered by vein or by mouth. The carbohydrate intake should be increased.

Fat soluble and water soluble vitamins must be supplied as concentrates for they will be inadequate if the food fat intake has been reduced to a minimum. Fatty foods with a high cholesterol content would infiltrate, especially in the absence of lipotropic agents. Proteins should be pushed to the limit of tolerance, if possible, 3 Gm per kilogram of

	METRIC	HOUSEHOLD MEASURE LEVEL	PROT	FATS	CARBO
<b>Breakfast</b>					
Boiled ham	100 Gm	3 x 3 x 1½	17	20	
Meat medium fat	100 Gm	½ cup	1		4
Vegetables 5 %	100 Gm	½ cup	2		9
Vegetables 10 %	100 Gm	½ cup	3		19
Vegetables 0 %	100 Gm	½ cup	2		10
Bread	30 Gm	1 slice		17	
Butter	20 Gm.	4 tsp		8	25
Desert (cake pudding)	1 serv		6	8	10
Milk	200 c.c.	¾ cup			
Coffee					
Cream 0 %	30 c.c.	2 Tbs.	1	6	1
Sugar	12 Gm	2 tsp +			12
2 P.M.			12.5	0.5	8.5
Brewers yeast	25 Gm	5 tsp	4.5	6	7.5
Milk	150 c.c.	¾ cup			12
S ga	12 Gm.	2 tsp +			18
3 P.M.					
Orange juice	200 c.c.	¾ cup			
<b>Supper</b>					
Soup (Julienne)	200 c.c.	¾ cup	4		4
Meat medium fat	100 Gm	½ cup scant	17	20	
Vegetable 5 % salad	100 Gm	½ cup scant	1		4
Vegetable 20 % salad	100 Gm	½ cup scant	3		19
Bread	30 Gm.	2 Tbs	2		15
Butter	20 Gm.	4 tsp		17	
Milk	200 c.c.	¾ cup	6	8	10
Fruit 13 %	100 Gm	½ cup scant			18
Tea					
Cream 0 %	30 c.c.	2 tbs.	1	6	1
Sugar	12 Gm	2 tsp +			12
7 P.M.			6	8	10
Milk	200 c.c.	¾ cup			
Grand Total			139	175	365
Calories 3,591					

Milk Nog Yeast is stirred thoroughly in cold milk and flavored with sugar, vanilla and nutmeg

### Acute Liver Damage—Parenchymatous Hepatitis Feeding (Comatose Jaundiced Cases)

#### High Protein Mixture by Duodenal Catheter

B

Milk skimmed	3 000 c.c.
Powered skimmed milk	180 Gm
Banana powdered	150 Gm.
Dextrose	300 Gm.
Vitamin A concentrates of cod liver oil	30 c.c.
Vitamin B, Brewers yeast	60 Gm.

Sig 200 c.c. every two hours, DAY and NIGHT by duodenal

#### Low Fat Low Lipoid, or Low Cholesterol Diet

Satisfactory evidence is available now that a very rich diet with a high intake of quantities of animal fats may be deleterious in certain individuals. Long term blood chemistry studies have shown that in most

as oranges, grapefruits, cantaloupes, melons, grapes, and all fresh fruits and frozen foods, and canned and dried fruits are acceptable Jello, jam, jelly, syrup, milk puddings, blancmange, and Junket made with out fats may be served as desserts Condiments should be kept to a minimum Skimmed milk, fresh or fermented milk, thickened with powder, may be added

## STANDARD DIETS FOR CIRRHOSIS PATIENTS

		METRIC	HOUSEHOLD MEASURE LEVEL	PROT	FAT%	CARBO
<b>Semiliquid Diet</b>						
7 A M	Milk	200 cc	$\frac{3}{4}$ cup	6	8	10
8 A M	Cereal (Pablum)	100 Gm	$\frac{1}{2}$ cup scant	2	1	11
	Sugar	12 Gm	2 tsp			12
	20 % Cream	30 cc	2 Tbs	1	6	1
	Eggs	2		13	10	
9 A M	Orange juice	200 cc	$\frac{3}{4}$ cup			18
10 A M	Eggnog					
	Milk	130 cc		45	6	75
	Egg	1		65	0	
	Sugar	10 Gm	2 tsp			10
	Brewers' yeast	25 Gm	5 tsp	125	05	85
11 A M	Cream soup	200 cc	$\frac{1}{4}$ cup	6	14	15
	Mashed potatoes	100 Gm	$\frac{1}{2}$ cup	2	6	15
	Butter	10 Gm	2 tsp		85	
	Purced vegetables	100 Gm	$\frac{1}{2}$ cup			9
	Orange juice	200 cc	$\frac{3}{4}$ cup			18
12 NOON	Cocomalt	200 cc	$\frac{3}{4}$ cup	6	10	20
2 P M	Eggnog	200 cc	$\frac{3}{4}$ cup	11	11	175
3 P M	Orange juice	200 cc	$\frac{3}{4}$ cup			18
4 P M	Cereal (Pablum)	100 Gm	$\frac{1}{2}$ cup	2	1	11
	Sugar	12 Gm	2 tsp +			12
	Cream 20 %	30 cc	2 Tbs	1	6	1
	Jello	100 Gm	$\frac{1}{2}$ cup	1		18
	Cream 20%	30 cc	2 Tbs	1	6	1
	Orange juice	200 cc	$\frac{3}{4}$ cup			18
5 P M	Cocomalt	200 cc	$\frac{3}{4}$ cup	6	10	20
6 P M	Eggnog	200 cc	$\frac{3}{4}$ cup	11	11	175
	Brewers' yeast	25 Gm	5 tsp	125	05	85
7 P M	Eggnog	200 cc	$\frac{3}{4}$ cup	11	11	175
<b>Grand Total</b>				116	131	315
Calories 2903						
<b>Solid Diet</b>						
<b>Breakfast</b>						
Fruit 18 %		1 serv				18
Coked cereal or		200 Gm	$\frac{1}{4}$ cup	4	2	20
Prepared cereal		30 Gm	$\frac{1}{4}$ cup			
Sugar on cereal		12 Gm	5 tsp +			12
Egg		2 only		13	10	
Milk		200 cc	$\frac{3}{4}$ cup	6	8	10
Toast		60 Gm	2 slices	4	1	30
Butter		20 Gm	4 tsp		17	
Coffee						
	Cream 20 %	30 cc	2 Tbs	1	6	1
	Sugar	12 Gm	2 tsp +			12
9 A M	Brewers' yeast	25 Gm	5 tsp	125	05	85
	Milk	150 cc	$\frac{1}{4}$ scant	45	6	75
	Sugar	12 Gm	2 tsp +			12

	METRIC	HOUSEHOLD MEASURE LEVEL	PROT	FATS	CARBO
Dinner			17	20	
Meat medium fat	100 Gm	3 x 3 x 1½	1		4
Vegetables 5 %	100 Gm	½ cup	2		9
Vegetables 10 %	100 Gm.	½ cup	3		19
Vegetables 20 %	100 Gm	½ cup	2		10
Bread	30 Gm	1 slice		17	
Butter	20 Gm.	4 tsp	5	8	25
Dessert (cake pudding)	1 serv		6	8	30
Milk	200 c c	¾ cup			
Coffee			1	6	1
Cream 10 %	30 c.c.	2 Tbs.			12
Sugar	12 Gm	2 tsp +			8.5
2 P M			12.5	8.5	
Brewers' yeast	25 Gm	5 tsp	4.5	6	7.5
Milk	150 c.c.	¾ cup			10
Sugar	12 Gm.	2 tsp +			18
3 P M					
Orange juice	100 c.c.	¾ cup			
Supper			4		4
Soup (Julienne)	200 c c	¾ cup	17	20	
Meat medium fat	100 Gm.	½ cup scant	1		4
Vegetable 5 % salad	100 Gm.	½ cup scant	3		19
Vegetable 20 % salad	100 Gm.	½ cup scant	2		15
Bread	30 Gm.	2 Tbs		17	
Butter	20 Gm.	4 t.p	6	8	10
Milk	200 c.c.	¾ cup			18
Fruit 18 %	100 Gm	½ cup scant			
Tea			1	6	1
Cream 10 %	30 c.c.	2 tbs			12
Sugar	10 Gm.	2 tsp +			10
1 P M			6	8	
Milk	100 c.c.	¾ cup			
Grand Total			139	175	365
Calories 3,591					

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### Acute Liver Damage—Parenchymatous Hepatitis Feeding (Comatose Jaundiced Cases)

#### High Protein Mixture by Duodenal Catheter

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Milk skimmed	3 000 c c
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Banana powder 1	150 Gm
Dextrose	300 Gm
Vitamin A concentrates of cod liver oil	30 c c
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q g 100 c c every two hours DAY and NIGHT by duodenal

#### Low Fat Low Lipoid or Low Cholesterol Diet

Satisfactory evidence is available now, that a very rich diet with a high intake of quantities of animal fats may be deleterious in certain individuals. Long term blood chemistry studies have shown that in most

patients the blood cholesterol is increased by a high cholesterol diet and decreased by a low cholesterol diet. A transitory lipemia with high levels of blood lipids develops after a heavy fat meal and lasts for a variable period of time. Free cholesterol and esters are soluble in the blood plasma.

Hypocholesterolemia depends upon dietary habits of the race, the diathesis, the individual's own make up, his ability to handle fats, and his general metabolic states. We have no knowledge of the enzyme or hormonal system that effects and controls the metabolism of the lipid and of cholesterol. We know that cholesterol may be synthesized from acetates occurring in food metabolism of fats and also of glucose and of proteins. If hypercholesterolemia is primarily of this endogenous origin the restrictions in that of exogenous origin would be ineffective. It is possible, however, that a heavy endogenous cholesterol metabolism may be overwhelmed by an added excessive intake of exogenous cholesterol.

It must be admitted that in atherosclerosis the high cholesterol level in the blood is by no means the whole story, but that other factors play very important roles particularly the permeability of the intima. This permeability may be increased by pressures in strategic areas by turbulence in the blood stream. Subintimal atherosclerosis is to be noted in patients with hypertension, nephritis, arteriovenous aneurysm endocrine disorders, poorly controlled diabetes mellitus myxedema, nephrosis and chronic cystic pancreatitis. In Niemann Pick disease Hand Schuller Christian disease acromegaly and Cushing's syndrome widespread atherosclerosis is usually found prematurely. It has been shown in our series and in those of others that there is a high incidence of hypercholesterolemia in patients with coronary arteriosclerosis especially in the younger age groups.

In such individuals it seems well to restrict the intake of foods rich in cholesterol. At present the means of rendering the absorption by the intestinal esterification and our means of decholesterolizing by lipotropic agents in the presence of high blood levels are still in the experimental stage.

In a low cholesterol diet foods rich in lipoids animal and dairy fat as eggs, butter, cream rich milk ice cream pork bacon sausage and fat rich fish such as salmon mackerel shad pompano and bluefish should be avoided. Vegetable fats containing phytocholesterols are usually better tolerated, as in avocado and olives. Those vegetable oils which are made up of unsaturated fatty acids with larger proportions of particularly linoleic and linoleic acids as olive oil corn and peanut oil are preferable substitutes. These oils should be limited and those as cottonseed oil, which have been hydrogenated as in Crisco and in margarine, and those containing saturated fatty acids seem almost com-

parable to the animal fats. Buttermilk made from skimmed milk is fat free. Green beans, peas and vegetables are to be cooked without adding fat. Dry vegetables, cereals, starches and sugar are foods of low cholesterol content and of low potential and may be used freely in the low fat low cholesterol diets.

## Cholesterol Content of Various Foods

HIGH AND MEDIUM CONTENT	MG /100 G	LOW CONTENT	MG /100 G
Brains beef	3700.0	Flour wheat	24.60
Kidney mutton	3400.0	Cornmeal	20.4
Pancreas calf	3120.0	Olive ripe	18.0
Salmon roe Tuna	2200.0	Beans green fresh	17.8
Egg yolk hen	2150.0	Melons	16.7
Chicken with skin	570.0	Milk cows skim	11.300
Egg whole hen	490.0	Pears	14.0
Lard suet	350.0	Oranges	13.6
Corn black	295.0	Asparagus	10.3
Beef muscle dried	230.0	Strawberries	9.1
Butter	20.0	Bananas	7.7
Oysters	14.0	Apples	7.4
Liver beef	140.0	Grapes white	6.3
Shrimp large	116.2	Peaches	6.2
Bacon fat	108.0	Spinach	5.5
Sardines	83.2	Apricots	3.2
Cream	83.0	Potatoes	2.6
Leaf fresh	60.0	Lettuce	2.3
Cream heavy	61.5	Cocoa	1.6
Chocolate	55.6	Carrots	1.2
Sausage	50.8	Sauerkraut	1.0
Beef very lean	37.0	Celery	0.8
Ham very lean	33.5	Beets	0.5
Fillet fish dry	21.0	Tomatoes	0.4
		Egg, white, hen	0.0

## Fat Content of Some Common Foodstuffs

100.80 %	40.30 %
Butter	Cheese plain or cream
Olive oil	Mutton medium fat
Lard	Fish roe
Cotton seed oil	Cream heavy
Suet	Potato chips
	Pork medium
	Peanuts
80.60 %	
Brains pancreas thymus sweetbreads	
Walnuts	
Bacon medium fat	
Salt pork	
	30.20 %
60.40	Beef medium fat
Eggs yolk	Tongue canned
Bacon lean	Ham lean
Ham fat	Lamb
	Steak porterhouse
	Salmon Tuna Pom
	pano
	Herring
	Mackerel salt
	Pickered Haddock

Pork, fat	Pecans	Cream light	Flounder, Trout
Lamb, fat	Beef, liver, kidney	Gelatin	Olives
Pork, loin chops		Hominy grits	Turkey
Sausage, pork			

20 10 %

Beef, sirloin, heart, round	Eggs
Capon chicken breast	Lamb roast
Codfish, herring, smelt	Salmon canned and fresh
Chicken broilers (muscle)	
Cracker, plain	

10 1 %

Red snapper, drumfish, porgy, shad, shellfish, scallops clams	Pears cooked
Bread white brown, cornbread, mush, whole wheat, rye	Potatoes
Apples	Milk, condensed
Carrots	Macaroni
Cheese cottage	Oats, rolled
Fruits, oranges, peaches, pears	Beans, string
	Beet, cooked
	Buttermilk, skimmed
	Egg white

With digestive disturbances salads, raw fruits and raw vegetables should be omitted and all vegetables should be purged

Some physicians allow vegetable fats, but I restrict all fats very sharply

## Low Fat or Low Cholesterol Diet

### (A Practical Outline)

*Avoid all foods high in cholesterol animal fats and dairy products as egg yolk, mayonnaise, butter cream ice cream rich milk cheese except Swiss cheese spreads fat meats especially pork bacon sausage, duck goose grease gravies dressing, bran sweet breads kidney, and liver*

*Fish roe oily fish as tuna salmon mackerel sardine and pompano*

*Foods prepared with eggs butter lard or goose grease chicken fats, creamed soups, custards, suet puddings fine rich, chocolate candies pastries cake pies*

*Vegetable fats condiments pepper pices salt succ citric and acetic acids and rich dressings are to be used sparingly Keep total calorie intake low not over 30 calories per kilogram (15 cal per pound) but proteins high at least 15 Gm per kilogram (0.8 Gm per pound)*

### BREAKFAST

*Fruit Applesauce baked apple, grapefruit melon orange peaches pears stewed or canned apricots stewed prunes, strawberries or other fresh fruit fruit juices, and unsipped tomato juice*

*Cereal Small dish of cooked or precooked cereal with skimmed milk An added tablespoonful of uncooked wheat germ is advantageous Oatmeal gruel made of rolled oats 40 Gm skimmed milk 200 c c*

*Proteins Broiled lean Canadian bacon with fat removed, a meat patty or album, as whites of eggs, should be cooked*

*Bread A slice of toasted dark bread with a little vegetable margarine margarine, jam No hot bread muffins, doughnuts, cookies, pancakes or waffles*

**Beverage** One pint of skimmed milk or natural buttermilk or a small cup of tea coffee or coffee substitute without cream, table spoonful of skimmed milk Small cup of cocoa tomato vegetable or fruit juices

## LUNCHEON AND DINNER

**Soups** A portion of consommé or bouillon, chicken tomato or mixed vegetable soup or clear meat broth once daily No creamed soups or fats

**Salads** Lettuce or any raw vegetable with gelatin or aspic, fresh or stewed fruits, and cottage cheese or French (no oil) or liquid petrolatum dressing oil which should be fortified with carotene or vitamin A concentrate

**No Oil French Dressing** Soften 1 t p plain gelatin in 1 Tbs cold water Dissolve mixture in  $\frac{1}{4}$  cup hot water adding 3 Tbs sugar and  $\frac{1}{4}$  tsp salt When cooled add  $\frac{1}{4}$  cup fresh lemon juice 1 t p dry mustard  $\frac{1}{2}$  tsp paprika + peppers, chopped onion garlic catsup or seasonings to taste Shake well Chill

**Bread** As above Soybean and whole wheat breads are preferable

**Oleomargarine** made entirely of vegetable oils may be used sparingly Lister's Spread is preferable

**Proteins Meats** Poultry fish None if patient can be a vegetarian otherwise, one small portion (175 Gm) daily of lean meat or fowl with skin removed, lightly broiled with the minimum of oleomargarine so that interior remains uncooked In order of preference beef lean round sirloin or roast chicken broiled roasted or boiled leg of veal or of lamb pork ham only very lean. Remove all visible fat Meat extractives without fat may be used but no gravies should be prepared

Fowl with skin removed chicken and turkey best preferable

**Fish** as red snapper trout flounder drum fish sea bass sheep head shrimp and small oysters are good substitutes

**Vegetables** At least 2 cooked without butter bacon salt pork or salad dressing Eggplant (boiled and seasoned or as greenless paricakes) artichokes and soybean meal are supposed to have decholesterizing activity Green and yellow vegetables are best eaten fresh and raw in salads as beet tops cabbage cauliflower carrots chard mustard kale celery dandelion turnips parsley parsnips spinach Swiss chard water cress tomatoes asparagus broccoli mushrooms onions starchy vegetables are boiled as hominy grits potatoes rice spaghetti macaroni dry beans and peas and should be prepared without fats as butter and served without gravy

**Desserts** Fruits fresh and stewed fruitwafers and sherbets or ices made with egg whites and gelatin egg white kisses or meringues tapioca or cornstarch pudding with skimmed milk no cream butter or eggs

Angel food cake jams jellies marmalade honey molasses maple syrup and sugars stick candy as desired if patient is not suffering from diabetes mellitus

## LOW FAT ICE OR SHERBERT

1 package of Knox gelatin in  $\frac{1}{2}$  cup water

1 cup water and 1 cup sugar heated to dissolve sugar Add gelatin and heat to dissolve

2 egg whites beaten stiff Add  $\frac{1}{2}$  cup white Karo

4 cups of orange juice

Combine all and freeze

Any fruit or fruit juice may be added or a light cocoa may be made using skimmed milk



Pork, fat	Pecans	Cream, light	Flounder, Trout
Lamb, fat	Beef, liver, kidney	Gelatin	Olives
Pork, loin chops		Hominy grits	Turkey
Sausage, pork			

20 10 %

Beef, sirloin, heart round	Eggs
Capon, chicken breast	Lamb roast
Codfish, herring, smelt	Salmon canned and fresh
Chicken, broilers (muscle)	
Cracker, plain	

10 1 %

Red snapper, drumfish, porgy shad, shellfish, scallops, clams	Peas cooked
Bread, white, brown, cornbread, mush, whole wheat, rye	Potatoes
Apples	Milk condensed
Carrots	Macaroni
Cheese, cottage	Oats rolled
Fruits oranges peaches, pears	Beans, string
	Beets, cooked
	Buttermilk skimmed
	Egg white

With digestive disturbances, salads raw fruits and raw vegetables should be omitted and all vegetables should be puréed

Some physicians allow vegetable fats but I restrict all fats very sharply

## Low Fat or Low Cholesterol Diet

### (A Practical Outline)

*Avoid all foods high in cholesterol animal fats and dairy products as egg yolks, mayonnaise butter cream ice cream, rich milk, cheese except Swiss cheese spreads, fat meats, especially pork bacon, sausage, duck goose grease gravies dressing bran, sweet breads kidney and liver*

*Fish roe oily fish as tuna salmon, mackerel sardines and pompano*

*Foods prepared with eggs butter, lard or goose grease chicken fats creamed soups custards, suet puddings fine rich chocolate candies pastries cake pies*

*Vegetable fats, condiments pepper pickle salt sauce citric and acetic acids, and rich dressings are to be used sparingly. Keep total calorie intake low not over 30 calories per kilogram (15 cal per pound) but proteins high at least 1.5 Gm per kilogram (0.8 Gm per pound)*

### BREAKFAST

*Fruit* Applesauce baked apple grapefruit melon orange peaches pears stewed or canned apricots, stewed prunes, strawberries or other fresh fruit fruit juice and unspiced tomato juice

*Cereal* Small dish of cooked or precooked cereal with skimmed milk. An added tablespoonful of uncooked wheat germ is advantageous. Oatmeal gruel made of rolled oats 40 Gm skimmed milk 200 cc

*Proteins* Broiled lean Canadian bacon with fat removed a meat patty or albumin as whites of eggs should be cooked

*Bread* A slice of toasted dark bread with a little vegetable margarine, marmalade, jam. No hot bread muffins, doughnuts, cookies, pancakes, or waffles

- B Whole wheat and graham breads graham crackers, plain hard cookies  
 C Cooked vegetables without serving Any that are listed in Group I, also squash, celery okra hominy Butter but no fat may be added  
 D Fruit juices—orange juice, lemonade grape juice pineapple juice grapefruit juice tomato juice Very ripe bananas  
 E Cooked fruits (without peel or seed unless very soft should be pureed), baked apple apple-sauce stewed peaches stewed apricots stewed prunes canned peaches canned plums canned California white cherries  
 F Meats Beef lamb veal, or liver may be roasted baked, or boiled

### Group III

- A Cooked vegetables Cauliflower broccoli Brussels sprouts kale artichokes onions rapidly cooked tender cabbage very tender corn tomatoes sauerkraut eggplant pumpkin leek sweet potato baked bean mushrooms  
 B Raw vegetables Tomatoes lettuce slaw celery  
 C Raw fruits Very ripe pears peaches watermelon cantaloupe honeydew melon muskmelon orange grapefruit cherries plums  
 D Meat loaf & scrambled eggs  
 E Hot breads Honey Syrup  
 F Plain vanilla or chocolate ice cream eaten slowly Malted milk Fried eggs  
 G Olives dates figs plain hard candy grapes  
 EAT SLOWLY CHEW FOOD WELL REST AFTER EATING

## General Bland Diet Constituents

Foods selected for a Bland Diet should be selected from the following

Soup (strained) Mutton chicken beef rice, tapioca barley creamed vegetable

Meat Lamb chop boiled or broiled beef broiled steak roast beef or lamb broiled broiled or roasted chicken turkey or squash—scraped or minced to avoid long fibers

Fish Broiled baked or boiled

Milk or Milk Foods In all forms including custards butter and cheese

Eggs Raw soft cooked scrambled poached or as egg-nog

Vegetables Strained finely chopped or pureed cooked vegetables, i.e. asparagus spinach peas string beans lima beans beets carrots potatoes

Cereals Cream of Wheat Farina strained oatmeal wheat germ cereal Wheatena or Ralston rice cornstarch tapioca macaroni spaghetti, no less than day old wheat bread, toast Avoid bran and most whole wheat cereals

Fruits Bananas Milky acid fruits i.e. cooked pears peaches apples Not seeded fruits

Desserts Custards plain or with toppings blancmange custards simple puddings sponge or angel cake

Beverages Milk malted milk weak cocoa tea or coffee, Postum with only a small amount of cream

Avoid Salads all raw or coarsely cooked vegetables fatty or fried foods meat sauces pickle highly seasoned foods condiments raw fruits and fruit juices which are strongly acid hot breads long fiber meat strong beverages and pastries preserves

## Graduated Bland Diet for Irritable G I Tract

Articles of diet are arranged below in groups. At the onset of treatment only those items indicated in the instructions are to be eaten, omitting all others, also omit anything crossed out. As digestive function improves, increase the variety of the diet by adding further groups of foods from time to time. No restricted diet such as this should be followed indefinitely without returning regularly for a physician's observation and instructions.

The diet of every patient must be adjusted to suit the individual. If found by experience that any of the foods listed below does not agree, omit such foods, unless ordered otherwise. Remember it is a menu to choose from and it is not necessary to eat all of it.

### Foods not to be eaten

- 1 Avoid all fried or greasy food, and gravies
- 2 Avoid all canned, pre-cooked or smoked meat or fish
- 3 Avoid raw apples, berries and similar raw, coarse or irritating fruits
- 4 Avoid all spices, seasoning sauces and dressings
- 5 Avoid pastries, rich candied jams
- 6 Avoid coarse and scratchy foods such as pickles, onions, radishes, cucumbers, pork and game
- 7 Avoid alcoholic and carbonated drinks and coffee and tea unless specifically permitted by the doctor
- 8 Avoid between meal eating unless specifically instructed by the doctor
- 9 Avoid very cold and very hot foods or drinks

### Foods that may be eaten

#### Group I

- A Well done cooked cereals, as Wheatena, oatmeal, Cream of Wheat, Farina, mush, grits
- B Sweet milk, butter milk, Postum, Ovaltine, cocoa, Kaffee Hag, Sanka, water
- C White bread (preferably slightly dried, or toasted), crackers, butter, oleo margarine, milk toast, macaroni, baked noodles, rice
- D Cream soups (any except onion soup or tomato soup), Oyster soup, Gruels
- E Cooked vegetables—should be puréed (put through a sieve), green or wax or butter beans, green peas, beets, carrots, turnips (young), spinach, tender asparagus tips, potatoes (mashed)
- F Meat broth (not greasy), Cottage cheese, Cream cheese, Small amount of chicken or fish broiled, roasted, or creamed once a day, Crisp bacon, eggs poached or boiled, Junket
- G Desserts, Puddings (such as tapioca, rice, cornstarch), Bavarian cream, Jello (plain), jelly, chocolate russe, prune whip, plain custard, blancmange, canned pears

#### Group II

- A. Uncooked cereals of the prepared type such as cornflakes, rice flakes, puffed rice and wheat. But not the very coarse "all bran" or "40 % bran" type or other very scratchy types

tion may be continued for two months. After this time the bacillus acidophilus preparation may be stopped but the patient should continue to take some lactose with his meals indefinitely.

### Mucous Colitis Cellulose Diet

#### AFTER ELIMINATION DIETS HAVE DETERMINED FOOD ALLERGIES

7 A.M.	Hot water $\frac{1}{2}$ pint with apples orange bananas figs grapes strawberries gooseberries etc. in season
8 30 A.M.	Tea coffee or cocoa made with milk one cup Fat bacon fish or eggs Brown whole meal bread (toast), 2 slices. Butter honey raspberry strawberry or black currant jam
Lunch	Egg savory vegetable dish e.g. baked tomatoes beans and tomatoes artichokes salad green or with boiled beetroots and celery Small allowance of water brown bread
4 30 P.M.	Tea with brown bread and butter Vegetable soup celery turnip onion carrot broth, bean Fish (fried or steamed) with bread or toast. Vegetable dish as at lunch Fruit (stewed) made into fritters or soufflé with cream Water to drink
9 P.M.	Glasses of hot water rusk or whole meal biscuit

Every food to which the patient is sensitive should be eliminated from the dietary and an appropriate substitution made.

### Sprue Low Fat Diet

Pratt used Schmidt's diet except in cases of sprue where a strict milk diet seemed better borne.

The following is exactly as given by Schmidt

Mornings	Milk 500 c.c. zwieback 50 Gm.
Forenoons	Oatmeal gruel (rolled oats 50 Gm. butter 10 Gm. milk 200 Gm., 1 egg and water 300 Gm. and salted to taste strain)
Noon	100 Gm. finely chopped roast beef (raw weight) lightly broiled so that the interior remains uncooked
Afternoon	As mornings
Evenings	As forenoon

Analysis of this diet	Protein	Fa	CHO	Calories
According to Schmidt	102	111	151	2234
Harlet and Goodbody	118	105	204	2297
Pratt	102	13	160	224

This diet is given for three days

To mark off the stools carmine 0.3 Gm. is taken with the first meal. The feeding period ended give on the following morning charcoal with

### Dietary Regime for Distention

*Part I*—A sharp restriction of the carbohydrate intake is to be followed for two to six weeks

The following foods are to be totally excluded from the diet potato, rice, root vegetables, dried beans, peas lentels, cabbage, cauliflower, Brussels sprouts, broccoli, peppers cucumbers, onions, garlic pickles, spices of all kinds and sweet milk

Green vegetables are to be eaten only when cooked and pureed Fruit must be taken either cooked or in the form of juices

The patient is allowed two tablespoonsful of macaroni or a well cooked cereal and one slice of white toasted bread with each meal There is no restriction on fats and nuts, except for peanuts

Ordinary amounts of proteins as eggs fish and cheese especially in the latter form are desirable Cottage cheese and gelatin are advised The gelatin is to be served in salads or with a meat or in the form of desserts

Fermented milk, especially buttermilk and acidophilus milk if it is available should be taken two or three times a day At first the symptoms may be slightly exaggerated and it is often advisable to postpone its use for a week or two and then begin with small amounts gradually increasing up to 1 quart per day

In addition to this colloidal kaolin or Fuller's earth is prescribed in doses of 8 to 15 Gm three or four times a day between meals with mineral oil The mineral oil is necessary under such circumstances to keep the bowels moving and if any difficulty is experienced an oil enema should be used Natural laxatives as Mucilose may act better

*Part II*—During the second period beginning at the third or fourth week starches sugars and well cooked cereals are gradually increased in the diet until normal proportions are reached The administration of fermented milk and kaolin is continued as previously The regime should be carried out for three or four weeks until the patient is free of symptoms

*Part III*—During the third period an attempt is made at bacterial implantation of acidophilus organisms The patient is put on the fermented milk or if this is not well tolerated 4 cc or a teaspoonful of the standard culture of bacillus acidophilus a Sharp & Dohme preparation, three times a day The patient is also instructed to begin the use of lactose or lactodextrin at mealtimes starting with a teaspoonful and working up to a tablespoonful three times a day Kaolin medica-

are obtainable from fresh vegetables particularly green ones as lettuce endive radishes onions spinach, celery mushrooms green beans cabbage, sauerkraut Brussels sprouts cucumbers pickles broccoli cauliflower, broiled okra eggplant and squash

(See section on Neurology and Psychiatry for further discussion on Obesity )

### General Directions and Suggestions for Those Overweight

- 1 Avoid all fats fat meats as pork butter cream, oils mayonnaise rich foods with shortening
- 2 Avoid all sugar and candies Saccharin may be used as a substitute as Crystalline or Sucryl
- 3 Add no salt to the food which should be prepared as salt poor
- 4 Do not eat between meals If you get hungry eat a piece of celery or lettuce drink water or eat an apple
- 5 Do not drink water with meals but between meals on arising and on going to bed Mineral water is desirable
- 6 Exercise with discretion increase gradually and indulge only until fatigued not exhausted

### Reduction Diet

*Protein*—Two thirds gram per kilogram of normal body weight for adults for children  $1\frac{1}{2}$  grams

*Calories*—Not more than  $\frac{1}{2}$  the normal requirement—to be obtained from bulk vegetables and fresh or unsweetened cooked fruits

#### Breakfast

Fruit without sugar

Light cereal—puffed wheat puffed rice or cornflakes with milk no sugar

$\frac{1}{2}$  slice unbuttered toast

Coffee without cream sweetened with saccharin if desired

#### Dinner

Clear soup

Lean meat—chicken or fish

Vegetables—two large servings either hot or as salad with vinegar

Tea Water

#### Supper

Cold meat (lean) or eggs

Vegetables—1 large serving

Fresh fruit or fresh dessert sweetened with saccharin if desired

Tea Milk

### Continental Reduction Diet

Three to five mutton days two vegetable days and two mixed days with restriction of fats and carbohydrates

a breakfast consisting wholly of milk. The charcoal is best given in form of an emulsion  $\text{R Carbonis vegetabilis 15 Gm, Mucilage gummi arab 15 Gm, Aq menth pip 60 cc}$ . Of this, a 3 cc dose is taken. After beginning of the test diet, the first stool colored red with carmine is saved, and collections continued until charcoal appears. The first stool colored with charcoal is rejected. The feces of a normal person taking the Schmidt diet has a uniform consistence and a light brown color.

Study the weight of the stools, dry, and determine the recovery in the dried feces of nitrogen, fat, and CHO, in terms of % of the amount of feces.

**Fat** The amount lost in the feces ranged from 2.1 % in patient with normal digestion to 66.2 % in a man with obstruction of the common bile and pancreatic ducts. In the cases of steatorrhea with two exceptions, 24 % or more of the fat in the food was excreted in the feces.

**Nitrogen** In obstruction of the pancreatic ducts there was N loss in the stools indicating faulty digestion and absorption of proteins. When fat loss was due to absence of bile from the intestine the absorption of nitrogen remained normal. In sprue, the absorption of N is usually normal but may be disturbed.

**CHO** Was normally utilized in all cases of steatorrhea studied. The micro examination of fresh stool for fat may be misleading. The determination of percentage of N is of no value, it is necessary to know the percentage loss of the amount of N fed.

#### References

- Pratt J H. A Study of Steatorrhea, *Am J M Sc* 187: 222, 1934.  
Schmidt, Ad. The Chemical Investigation of Gastric and Intestinal Diseases by the Aid of Test Meals. London, 1906, p. 96.

### Dietetic Management in Obesity

The dietetic management of obese patients consists primarily of changing bad eating habits. The craving for carbohydrates which is generally present must be curbed. Diets rich in fats and carbohydrates are the chief cause of obesity. Glandular dysfunctions are only very rarely important factors. If hypothyroidism plays a role thyroid therapy may be helpful, but only when the metabolism is quite low.

In general the food value of the diet must be kept low but it must be well balanced, high in protein, low in carbohydrate and very low in fat. Very lean meat, fish, fowl with all the skin removed and all the fat removed, egg white, dry cottage cheese, gelatin, skimmed milk and buttermilk make up the main part of the diet. Minerals and vitamins

## Breakfast

Fruit juice Cooked cereal with crystalline and whole milk One slice of toast  
Large glass of water or milk

## Lunch

Clear or mild soup (no grease)

Lean meat or fish—baked or broiled lamb mutton beef chicken squab or lamb  
chops or whites of two eggs or 1 tablespoonful of dried beans or peas or  
cheese (portion the size of three fingers) or 3 tablespoonfuls of cottage  
cheese

Green vegetables 5 tablespoonfuls

Vegetables or fruit salad—small portion with mayonnaise made with mineral oil  
steamed or fresh fruit two tablespoonfuls or custard, gelatin, blancmange, sage  
cornstarch prune whip tapioca 2 tablespoons Sweeten with crystalline  
(sodium saccharin)

## Dinner

Fruit or vegetable salad—2 crackers Whole milk  $\frac{1}{2}$  pint or  $\frac{1}{4}$  pint of fruit  
juice If hungry between meals eat a heart of celery or drink tomato  
juice

Calories 900 to 1100

On arising  $\frac{1}{2}$  hour before breakfast take either lemon in water with crystalline (use  
no or very little sugar) or berries or baked apple or oranges No sugar or cream  
with the fruit

## Breakfast

- 1 Whole wheat bread or rolls or crackers butter Postum or buttermilk or sour  
milk
- 2 Bran biscuits or cornbread butter honey Postum or buttermilk and Postum  
or milk
- 3 Crushed banana and bran mixel and baked whole wheat bread butter and  
Postum

## Lunch

- 1 Baked fish baked potato creamed vegetable lettuce salad no bread  
Broiled sweetbread combination salad either baked potato or bread vegeta-  
bles
- 3 Roast chicken combination salad creamed corn or carrots whole wheat bread
- 4 Fish pot roast or chowder fresh tomato salad vegetables whole wheat bread
- 5 Baked potato butter buttermilk or sour milk whole wheat bread
- 6 Combination salad whole wheat bread butter buttermilk or sour milk or  
Postum

## Dinner

- 1 Vegetable soup one patty of ground meat or scrambled or boiled egg whole  
heart bread butter milk or Postum
- 2 Green pea soup vegetable salad or lettuce whole wheat bread butter milk or  
Postum
- 3 Lentil soup poached eggs creamed vegetables whole wheat bread butter, milk  
or Postum
- 4 Cream of celery soup cottage cheese whole wheat or corn bread butter milk  
or Postum



*Mutton or Lamb Days* (salt poor preparation total 3 Gm NaCl) —

Before breakfast on arising, mineral water or 150 c.c. warm water with Glauber salt (1 tablespoonful)

	CALORIES
1 2 cups of tea or maté 3 zwiebacks (no bread)	80
If hungry between meals 1 apple may be taken	30
<i>Noon Meal</i>	
2 mutton cutlets broiled or grilled, 100 Gm with very little butter or lard used	200
1 2 slices pineapple (raw) if possible, or sugar free preserved pineapple, 100 Gm	40 80
1 plate 100 Gm greens, lettuce with lemon juice	15
1 cup tea (black or maté) No bread	15
<i>Afternoon</i>	
Mineral water or Glauber's salt 1 cup tea and 1 zwieback	30
If hungry between meals, 1 apple or equivalent fruit (no bananas)	30
<i>Dinner</i> as noon meal	255
Total	680 750

*Green Vegetable and Fruit Days* (salt poor preparation total 3 Gm NaCl) —

Before breakfast Kissingen mineral water or Glauber's salt

<i>Breakfast</i>	
1 2 cups tea or maté 3 zwieback	80
100 Gm fruit	60
<i>Noon</i>	
1 cup fat free bouillon 250 Gm fresh vegetables	150
Vegetables cooked in bouillon	
Green cauliflower green beans green peas spinach	
kale, turnips, carrots Mohren carrot asparagus, celery	
cabbage (red, green, rose) crushed cabbage and	
viper's grass	
100 Gm Greek lettuce with lemon juice	15
250 Gm fruit or sugar free stewed fruit or preserved	150
1 cup of tea or maté	
<i>Afternoon</i> Coffee without cream or sugar, 3 zwieback	80
<i>Dinner</i> as noon meal	315
Total	850

**Low Caloric Diet**

*Rest* — The patient should lie down for one hour after the noon meal

*Exercise* should consist of slow walking in the open air on level ground gradually increasing distances from a few feet up to half a mile

The following is suggested as a trial diet

FOODS	CALCIUM CONTENT	PHOSPHORUS CONTENT
<i>Meats</i>		
Steak (round fat)	0.008	0.172
Chicken (dark)	0.000	0.138
Turkey	0.008	0.212
Goose	0.010	
Liver	0.006	0.20
Ham	0.000	
<i>Vegetables</i>		
Tomatoes (fresh)	0.007	0.035
Rice (uncooked)	0.007	0.096
Yeast	0.019	1.10
<i>Cereals</i>		
Bread, regular	0.041	0.093
Crackers, Graham	0.060	0.111
Shredded Wheat	0.030	0.310
Farina	0.013	0.100
Macaroni	0.018	0.130

### Foods With High Calcium Content

The following foodstuffs are rich in calcium but contain very small amounts in relation to the amount required. Dairy products: cheese, milk, egg yolks and eggs. Fruits and vegetables: grapes, currants, gooseberries, strawberries, cherries, oranges, lemons, bananas, figs, pears, pine apple, citron, turnips, greens, lettuce, watercress, cauliflower, cabbage, asparagus, radishes, endive, spinach, kohlrabi, rhubarb, artichokes, carrots, peas, barley, lentils, beans, ripe olives, almonds, walnuts, peanuts, and paprika. Tortillas, beef, pork, oysters, fish, corn meal, oatmeal, barley, and wheat flour, rice, and potatoes contain calcium in decreasing order.

### Low Potassium Diet

<i>Breakfast</i>	APPROXIMATE MEASURE	WEIGHT GM.
Orange juice	One half glass	100
Cornflakes	One serving	10
Egg	One	50
Bread	Two slices	50
Butter	Two squares	20
Cream 40% fat	Short half cup	75
Coffee		
<i>Dinner</i>		
Beef tenderloin	One average serving	70
Potatoes (if rice boiled)	One average serving	100
Carrots	One small serving	25

(uncooked wt.)

mix well by stirring and set aside to cool. When cool and congealed on the surface of the water, break up the cake of butter on the surface. Put the butter through a fine mesh wire strainer and wash thoroughly with ice water until the wash water is clear.

**Low Calcium Bread** Take flour, 435 Gm, add water 360 cc, washed butter 10 Gm, sugar 10 Gm, yeast 5 Gm, and salt C.P. 5 gr. Mix to prepare the dough in quick fashion. Bake at 430° F. for 30 minutes, then decrease the temperature to 380° and bake for 15 minutes more.

**Low Calcium Fudge** Mix sugar 200 Gm, water 150 Gm, chocolate 10 Gm and washed butter 10 Gm. Cook at a temperature of 240° F.

### Low Calcium Test Diet

	CALCIUM GM./100 GM.	PHOSPHORUS GM./100 GM.	AMOUNT GM.
<i>Breakfast</i>			
Bacon (broiled)	0.030	0.096	30
Low calcium bread	0.011	0.082	60
Butter (washed)	0.000	Trace	20
Potato (steamed)	0.011	0.0475	150
Apple (raw whole)	0.010	0.0120	100
Sugar (granulated)	0.000		
Coffee			
<i>Dinner</i>			
Steak (round lean)	0.008	0.172	100
Potato (steamed)	0.011	0.475	100
Corn (canned)	0.005	0.043	100
Low calcium bread	0.011	0.082	60
Butter (washed)	0.000		15
Banana (sliced)	0.007	0.043	100
Sugar	0.000		
Tea			200
<i>Mid-afternoon</i>			
Ginger ale	0.0005	0.001	200
<i>Supper</i>			
Chicken (white meat)	0.016	0.164	45
Rice (cooked)	0.006	0.075	75
Tomatoes (canned)	0.005	0.042	100
Low calcium bread	0.011	0.082	60
Butter (washed)	0.000	Trace	15
Apple (cooked)	0.100	0.012	100
Honey	0.004	0.018	30
Sugar	0.000		30
Tea, mineral oil laxative			200

Total Calcium Content Calculated 0.113, Actual 0.105 Calories  
2,345, Phosphorus Content Calculated 0.781, Actual 0.772  
Sodium Salt 3.05

## DIETETIC METHODS

	CALCIUM CONTENT	PHOSPHORUS CONTENT
<i>Meats</i>		
Steak (round fat)	0 008	0 172
Chicken (dark)	0 000	0 158
Turkey	0 008	0 212
Goose	0 012	
Liver	0 006	0 270
Ham	0 0 2	
<i>Vegetables</i>		
Tomatoes (fire b)	0 007	0 000
Rice (uncooked)	0 007	0 096
Veget	0 019	1 170
<i>Cereals</i>		
Bread, regular	0 041	0 093
Crispers Graham	0 060	0 111
Shredded Wheat	0 030	0 310
Farina	0 013	0 100
Maccaroni	0 018	0 130

## Foods With High Calcium Content

The following foodstuffs are rich in calcium but contain very small amounts in relation to the amount required Dairy products cheese milk e g yolks and eggs fruits and vegetables grapes currants goose berries strawberries cherries, oranges lemons bananas figs pears pine apple citron turnips greens lettuce watercress cauliflower cabbage asparagus radishes endive spinach kohlrabi rhubarb artichokes carrots peas barley lentils beans ripe olives almonds walnuts peanuts and paprika tortillas beef pork oysters fish corn meal oatmeal barley and wheat flour rice and potatoes contain calcium in decreasing order

## Low Potassium Diet

Per 1/2 pint	APPROXIMATE MEASURE	WEIGHT GM
Orange juice	One half glass	100
Cornflakes	One serving	15
E "	One	50
Bread	Two slices	50
Butter	Two squares	20
Cream, 40 % fat	Short half cup	"
Coffee		

## Dinner

Beef tenderloin	One average serving	70	(uncooked wt)
Potatoes (thru boiled)	One average serving	100	
Carrots	One small serving	25	

Celery	Two hearts	25
Grapefruit	Two sections	55
Bread	Two slices	50
Butter	Two and one half squares	25
Cream, 40 % fat	One tablespoonful	20
Tea or coffee		

*Supper*

Cheese	Two cubic inches	40
Rice (dry wt)	One average serving	25
Tomato	One half average serving	50
French dressing	One tablespoonful	15
Apple	Small, $\frac{3}{4}$ average serving	80
Bread	Two slices	50
Butter	Two and one half squares	25
Cream 40 % fat	One tablespoonful	20
Tea or coffee		

Potassium Content 16 Gm, Protein 57 Gm, Calories 2,350

In Addison's disease, besides a high sodium salt intake, potassium must not exceed 2 grams

See lists of foods according to the *Potassium Content*

Reference Mary Victor Sr Proc Staff Meet Mayo Clinic 12 424 432 July 7, 1937

### Low Potassium, High Sodium Diet

Reference Wilder, R M Kendall E C Snell A M, Kepler E J Rynearson E H, and Adams M Intake of Potassium and Important Consideration in Addison's Disease, Arch Int Med 59 367 Mar 1937

Potassium 16 Gm, Proteins 57 Gm Calories 2350

Food	GRAMS	APPROXIMATE MEASURE
<i>Breakfast</i>		
Orange juice	100	$\frac{1}{4}$ glass
Cornflakes	15	1 serving
Egg	50	One
Bread	50	2 slices
Butter	10	2 squares
Cream 40 % fat	75	$\frac{3}{8}$ cup
Coffee if desired		

*Dinner*

Beef tenderloin (wt uncooked)	70	1 average serving
Carrots	25	1 small serving
Potato thrice boiled	100	1 average serving
Celery	25	2 celery hearts
Grapefruit	55	4 sections
Bread	50	2 slices
Butter	25	2 $\frac{1}{2}$ squares
Cream, 40 % fat	20	1 tablespoonful
Tea or coffee if desired		

*Supper*

Rice (wt dry)	25	1 average serving
Cheese	40	2 cubic inches
Tomato	50	½ average serving
French dressing	15	1 tablespoon
Apple	80	¼ average size
Bread	50	2 slices
Butter	25	2½ squares
Cream 40 % fat	20	1 tablespoonful
Tea or coffee if desired		

Vegetables e.g. potato may be boiled in three changes of water and from 75 to 80 % of salts removed subsequently season with pure measured salt. High potassium containing foods are avoided.

### Foods With High Potassium Content

The following foods are rich in potassium and contain over 300 mg of K per 100 Gm. meats fowl fish of all kinds, potatoes sweet potatoes peas beans beets cabbage celery chard chestnuts spinach and other greens pumpkin squash parsnips dried fruits especially dates figs prunes banana pineapple nuts of all kinds wheat bran and molasses. Paprika meats potato peel olives and nuts make a high K diet.

#### References

Mary Victor Br. A Diet Restricted in Potassium J. Am. Dietetic Assn. 14  
59, 7, 1938

### Iron in Foodstuffs

#### HIGH SOURCES

Asparagus	Lettuce
Dried Beans	Liver
Beet Greens	Nuts
Bran	Oatmeal
Chard	Oysters
Dandelion Greens	Parsley
Eggs	Soy Beans
Heart	Turnip Greens
Kidney	Water Cress
Peas	Whole Wheat

#### GOOD SOURCES

Apricots	Eggs
Bananas	Fish
Green Beans	Goose
Beef	Lamb
Beets	Mushrooms
Blackberries	Parsnips
Broccoli	Potatoes
Brussels Sprouts	Prunes
Cheese	Raisins
Dates	Raspberries
Duck	Spinach
Endive	Tomatoes
	Turnips

#### LOW SOURCES

Cabbage	Green Peppers	Grapefruit
Carrots	Sweet Potatoes	Grapes
Cauliflower	Radishes	Lemons
Celery	Rhubarb	Oranges
Collards	Rutabagas	Peaches

Canned Corn  
 Yellow Corn  
 Cucumbers  
 Eggplant  
 Kale  
 Onions

Squash  
 Apples  
 Blueberries  
 Cherries  
 Cranberries  
 Currants

Pears  
 Pineapple  
 Plums  
 Strawberries

### Purin Free Diets in Gout

**GIVE** foods of Low Purin  
 Content as

#### *Dairy Products —*

Milk  
 Butter  
 Cheese { Cottage  
           { Cream or  
           { Swiss  
 Malted Milk  
 Eggs  
 Nuts

#### *Cereals —*

Cornflakes  
 Shredded Wheat  
 Cream of Wheat  
 Farina  
 Rice  
 Hominy

#### *Bread —*

Wheat (white)  
 Crackers (white)

#### *Vegetables —*

Lettuce  
 Cabbage  
 Eggplant  
 Cauliflower  
 Potatoes (small amounts)  
 Sweet potatoes  
 Corn  
 Okra  
 Brussels sprouts

#### *Soups —*

Purée  
 Cream

**AVOID** foods of High Purin  
 Content as

#### *Meats and Fish (given in order of purin content)*

Sweetbreads (Thymus or Pancreas)  
 Brains, Liver Kidney  
 Beef  
 Fish  
 Poultry  
 Veal  
 Pork  
 Mutton  
 Game

Oatmeal

Graham bread  
 Whole wheat bread  
 Bran bread

Peas  
 Beans  
 Asparagus  
 Onions  
 Mushrooms  
 Potatoes (large amounts)

Meat stocks  
 Extractives  
 Noodles  
 Macaroni

## Purin Free Diets in Gout—Cont'd

GIVE foods of Low Purin  
Content as

AVOID foods of High Purin  
Content as

series —

Pudding  
 Bread or cornstarch  
 Rice or tapioca  
 Custard or coconut pie  
 Gelatin preparations  
 Stewed or preserved fruits  
 Ice cream or Ices  
 Cake  
 (No chocolate or coffee flavors)

fruits —

All kinds

Beverages —

Decaffeinated Coffee (Kaffee Hag or Sanka)  
 (small amount)

Coffee  
 Tea  
 Cocoa

Milk

Fruit juices especially grape juice  
 cider

Carbonated waters

Use occasionally —

Chicken bacon lamb oyster crab  
 lobster whitefish haddock

## Arthritic Diets

According to Pemberton sharp curtailment of food intake often influences greatly for the better or relieves all symptoms in certain cases of arthritis not amenable to other therapy

Additional support for such therapy was found in the blood sugar studies which revealed a difficulty in the utilization of carbohydrates

The diets are based on the principle of a lowered intake Not only are the calories reduced but the proportions of the foodstuffs believed to be advantageous are provided.

The caloric value ranges from 1 650 cal to 2 100 cal Of these calories 10 % come from protein 50 to 60 % from fat and 30 to 40 % from carbohydrate thus providing a low caloric, high fat, restricted or relatively low carbohydrate and moderate protein diet

Reference Pemberton Ralph Arch Int Med. 20 335 April, 1920



## Diet No 1

MENUS ARRANGED BY MISS CECILE DAVIS, BARNES HOSPITAL  
Weight in Grams

FOOD	8 A M	12 NOON	6 P M	ORDER	PROTEIN 41		FAT 113		CARBOHYDRATE 115		CALORIES 1641
Grapefruit	100			100					6		
Cream	50			50	1	2	10		2		
Bacon	20			20	1	9	13	4		2	
Eggs	2			2	13	4	10	50			
Coffee											
Sugar	22			22					22		
Potatoes		75		75	1	6	7		13	5	
Steak		75		75	14	7	9	5			
Asparagus (c)		100		100	1	5		11	2	3	
Pears		100		100		3		3	18		
Butter		20		20		2	17				
Consommé			100	100	2	2					
Tomatoes (c)			25	25		4		5		8	
Cream			45	45	1	9	9				
Okra			100	100		7		1	2	9	
Tomatoes (f)			100	100		9		4	3	3	
Lettuce											
Oil			20	20			20				
Olives			100	100	1	4	21		3	5	
Orange Juice			100	100		8		2	11	6	
Lemon Juice			50	50					4	9	
Sugar			44	44					44		
					43	1	11	7	115		1643.5

(c) cooked. (f) fresh

## Diet No 2

Weight in Grams

FOOD	8 A M	12 NOON	6 P M	ORDER	PROTEIN 42		FAT 118		CARBOHYDRATE 119		CALORIES 1700
Grapefruit	100			100					6		
Bacon	20			20	1	9	13	5			
Cream	75			75	1	8	15		2	5	
Sugar	30			30					30		
Coffee											
Cantaloupe		100		100		6			7	2	
Consommé		200		200	4	4			8	6	
Beets		100		100	1	6		1			
Potatoes		75		75	1	9		01	13	5	
Steak		75		75	14	7	9	5			
Tomatoes (c)		100		100	1	2		2	3	5	
Butter		15		15		11	12	7			
Olives			100	100		8	20	2	8	5	
Oil			10	10			10				
Lettuce											
Tomatoes (f)			75	75		6					
Ham			75	75	11	2	20	3	2	3	
Bananas			100	100	1	3		1			
Cream			30	30		7	6	6	21		
Sugar			20	20					20		
					42	8	117	2	121	1	1710.4

Diet No 3  
Weight in Grams

FOOD	8 A.M.	1 <sup>st</sup> NOON	6 P.M.	ORDER	PROTEIN 51		FAT 118		CARBO HYDRATE 199		CALORIES 2031
					51	6	118	9	199	2	
Cantaloupe	100			100							
Cornflakes	75			75	1	1	3	9	7	2	
Eggs	11			11	13	4	10	5	70	0	
Egg albumin	35			35	4	3			4	5	
Cream	35			100	2	5	20	8	5		
Butter	100			10		1		5	10		
Sugar	10			10				2	10	5	
Bread	10			20	1	8		5	1		
Chicken	90			50	10	5	1	5	18		
Potato		50		100		2		1	5		
Butter		100		10		1	8		4	3	
Tomato (f)		10		100		9		2	10	5	
Bread		100		20	1	8			19	7	
Rice		90		25	2	5	10		4	5	
Cream		100		100	2	5	10	2	10	5	
Wheat		90		20	1	8	10	8			
Butter		98		28		2	3		73	3	
Apples		100		100	2	1					
Oranges		100		35	4	3					
Butter white					51	3	117	8	19		2034

Diet No 4  
Weight in Grams

FOOD	8 A.M.	1 <sup>st</sup> NOON	6 P.M.	ORDER	PROTEIN 44		FAT 118		CARBO HYDRATE 213		CALORIES 2090
					44	5	118	0	213	5	
Cream	100			100					4	5	
Sugar	10			10					10		
Butter	10			10			8	5	1		
Shredded Wheat	0			90	2	1		1	15	2	
Egg	1			1	6	7	10	5			
Bacon	5			5	2	4	16	8			
Orange	100			100			8	2	11	6	
Ham		50		50	8	1	19	8			
Sweet Potato		100		100	1	8		7	26	1	
Canned Corn		100		100			1	2	18	2	
Pasta		50		50	1	3	1	7	38		
Butter		18		18			15	3			
Sugar		20		0					10		
Cheese				10	10		8	3	5		
Macaroni				25	5	4		1	1	7	
Potato				100	100	2	2		1	18	
Cream				25	25		6	5	8	1	
Butter				15	15		10	7			
Bread				15	75	3	9		8	39	3
Sugar				5	5				5		
					4		118	8	214		2086

### Regime for Thromboangitis Obliterans

Diet high in *Calcium* and *Vitamins* has a favorable effect in general and on vasospasm Vitamin D preferably in conjunction with Vitamin A as in concentrates like Adex (6 tablets per day), should be given to patients not getting sunlight or ultraviolet

#### Required Foods—Optional or Allowable

##### Breakfast

Orange or grapefruit juice 4 6 ounces  
Fruits (raw or cooked)  
Wheat Germ (not to be cooked) 1 2 heaping Tbs  
Milk, 1 pint  
Coffee or tea with sugar and cream  
Bread, 1 slice with butter

##### Noon

Milk (sweet or buttermilk) 1 pint  
Broiled meats  
Fruits (raw or cooked)  
Vegetables (except potatoes)  
Corn, rice, spaghetti  
Cheese, any kind  
Salads—fruits and vegetables  
Bread, 1 slice with butter

##### Evening

Tomato or pineapple juice (canned) 4 6 ounces (not cocktail)  
Meat or fish  
Any food desired to make up calories

There should be 5 hour intervals between meals Nothing is to be eaten between meals

### Nephritic Diet Sheet

O'HARE AND WICKERS

Any combination of the foods listed below may be selected

Foods not listed below must not be taken

In Groups I and II there is a restriction in the total amount

The foods in those groups must be served in full or half portions

A full portion in Group I counts 1

A full portion in Group II counts 2

In Group III the quantity of each is not restricted, although you are urged to use discretion

Each unit contains 4 Gm of protein

YOUR TOTAL SCORE FOR THE DAY SHOULD BE-----

YOUR TOTAL AMOUNT OF FLUID SHOULD BE-----PINTS

Do not add salt or spices to the food after it has been cooked

## GROUP I

(Each full portion counts 1)

	Full Portion	Vegetables	Full Portion
Bread (white)	1 av slice	Baked beans	1 tbp
Bread (graham)	1 av slice	Lima beans	1½ tbp
Unseeded biscuit	5 crackers	Potato creamed	1 tbp
Shredded Wheat	1 biscuit	Potato mashed	2 tb p
		Potato boiled	1½ mel
		Green peas	2 tb p
		Canned corn	2½ tl p
Cereals		Onion boiled	3 tb p
Oatmeal	2 tbp	Macaroni	4½ tb p
Boiled rice	3 tb p	Squash boiled	6 tb p
Cornmeal mush	4 tl p	Orange	2 av size
Cream of Wheat	6 tb p	Grapefruit	1 small
Farina	6 tbp		

## GROUP II

(Each full portion counts 2)

	Full Portion	Meats	Full Portion
Milk	1 glass	Chicken roast	3 × 3 × ½
Eg	1 egg	Lamb chop broiled	2½ chop
Eg s (scrambled)	1½ tbp	Lamb roast	3 × 2½ × 1¼
Custard	3 tb p	Beef roast	3 × 2 × ¾
		Beef steak broiled	2 × 1 × 1
Fish			
Cod boiled	1 × 1 × 1¼		
Haddock boiled	1½ × 1 × 1		
Oysters	7 oysters		

## GROUP III

(No restriction)

Vegetables	Fruit	Miscellaneous
Turnips	Watermelon	Sugar Cornstarch
Carrots	Plums	Syrup Arrowroot
Cabbage	Pears	Candy Tapioca
String beans	Peaches	Honey Post Toastles
Cucumbers	Strawberries	Maple Maple Syrup
Cauliflower	Grapes	Sugar Olive Oil
		Butter

## Low Protein Diets

You may use any one of the following diets on any given day. The whole diet for that day must be taken exactly as indicated, no swapping of parts of diets being allowed. Please do not eat anything except what is included in your diet. No salt or spice is to be added after cooking. You are to take -- -- pints of fluid per day.

No 1		No 2		No 3		No 4	
$\frac{1}{2}$ Orange		$\frac{1}{2}$ Orange		$\frac{1}{2}$ Orange		$\frac{1}{2}$ Baked Apple	
3 h tbsp Crm of		3 h tbsp Oat		3 h tbsp Oat		3 h tbsp Oat	
Wheat		meal		meal		meal	
1 slice Bread		1 slice Toast		1 slice Toast		1 slice Toast	
$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$	
4 teasp Butter		1 cup Coffee		1 cup Coffee		1 cup Coffee	
1 cup Coffee		8 teasp Cream		8 teasp Cream		8 teasp Cream	
8 teasp Cream		5 teasp Sugar		5 teasp Sugar		5 teasp Sugar	
5 teasp Sugar		4 teasp Butter		4 teasp Butter		4 teasp Butter	
$\frac{1}{2}$ cup Cream Soup		$\frac{1}{2}$ cup Cream Soup		$\frac{1}{2}$ cup Cream Soup		$\frac{1}{2}$ cup Cream Soup	
1 Potato		1 Potato		1 Potato		1 Potato	
3 h tbsp Peas		3 h tbsp Carrots		1 Beet		2 h tbsp Squash	
1 slice Bread		1 slice Bread		1 slice Bread		1 slice Bread	
$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$	
4 teasp Butter		4 teasp Butter		4 teasp Butter		4 teasp Butter	
Blancmange		Spanish Cream		Blancmange		Fruit Salad	
$\frac{1}{2}$ cup Cream		$\frac{1}{2}$ cup Cream		$\frac{1}{2}$ gla s Cream		$\frac{1}{2}$ Apple	
1 teasp Sugar		1 tea p Sugar		1 teasp Sugar		$\frac{1}{2}$ Peach	
1 egg white		1 egg white		1 egg white		$\frac{1}{2}$ cup Cherries	
1 tea p Corn		1 tea p Gelatin		1 teasp Corn		av portion Let	
starch				starch		tuce	
1 cup Tea		1 cup Tea		4 teasp Cream		Mayonnaise	
8 teasp Cream		8 teasp Cream		1 cup Tea		1 cup Tea	
2 teasp Sugar		2 teasp Sugar		teasp Sugar		4 teasp Cream	
						teasp Sugar	
3 h tbsp Cabbage		2 h tbsp Maca		2 h tbsp Maca			
		roni		roni			
1 slice Bread		1 sm Tomato		1 med Tomato		3 h tbsp Peas	
$3 \times \frac{1}{2} \times \frac{1}{2}$							
4 teasp Butter		1 slice Bread		3 h teasp Apple		1 llice Bread	
		$3 \times \frac{1}{2} \times \frac{1}{2}$		sauce		$3 \times \frac{1}{2} \times \frac{1}{2}$	
3 h tbsp Apple		4 teasp Butter		1 cup Tea		4 teasp Butter	
sauce							
1 cup Tea		2 slices Pineapple		8 teasp Cream		1 sm Pear	
8 teasp Cream		1 cup Tea		5 teasp Sugar		2 h tbsp Maca	
						roni	
5 teasp Sugar		8 teasp Cream		1 slice Bread		1 cup Tea	
		5 teasp Sugar		$3 \times \frac{1}{2} \times \frac{1}{2}$		8 teasp Cream	
				4 teasp Butter		tea p Sugar	
No 5		No 6		No 7		No 8	
1 sm bunch		$\frac{1}{2}$ Orange		1 bunch Grapes		$\frac{1}{2}$ Orange	
Grapes							
3 h tbsp Oat		3 h tbsp Crm of		3 h tbsp Oat		3 h tbsp Oat	
meal		Wheat		meal		meal	
1 slice Bread		1 slice Toast		1 slice Toast		1 llice Toast	
$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$		$3 \times \frac{1}{2} \times \frac{1}{2}$	
4 teasp Butter		1 cup Coffee		4 teasp Butter		1 cup Coffee	
1 cup Coffee		$\frac{1}{2}$ glass Cream		1 cup Coffee		8 teasp Cream	
8 teasp Cream		5 teasp Sugar		8 teasp Cream		5 tea p Sugar	
5 teasp Sugar		4 teasp butter		1 teasp Sugar		4 t asp Butter	
1 Potato		$\frac{1}{4}$ cup Cream Soup		1 sweet Potato		$\frac{1}{2}$ cup Cream Soup	
1 ear Corn		1 av size ear of		1 ear Corn		1 Potato	
$\frac{1}{2}$ glass Cream		Corn		Fruit Salad		1 med Beet	
1 slice Bread		1 small Potato		av portion Let		1 slice Bread	
$3 \times \frac{1}{2} \times \frac{1}{2}$		1 slice Bread		tuce		$3 \times \frac{1}{2} \times \frac{1}{2}$	
4 teasp Butter		$3 \times \frac{1}{2} \times \frac{1}{2}$		$\frac{1}{2}$ Orange		Custard	
1 sm Baked		4 teasp Butter		$\frac{1}{2}$ cup Cherries		$\frac{1}{2}$ glass Cream	
Apple		Spanish Cream		1 m Peach		1 teasp Sugar	
1 cup Tea		$\frac{1}{4}$ glass Cream		Mayonnaise		1 Egg yolk	
4 teasp Cream		1 teasp Sugar		1 cup Tea		1 cup Tea	
2 teasp Sugar		1 egg white		2 teasp Cream		4 teasp Butter	
		$\frac{1}{4}$ teasp Gelatin		10 teasp Sugar			
		1 cup Tea		4 teasp Butter			
2 h tbsp Maca		1 tea p Cream					
roni		2 tea p Sugar					
1 sm Tomato							
1 slice Bread		3 h tbsp String		2 h tbsp Maca		1 Onion	
$3 \times \frac{1}{2} \times \frac{1}{2}$		Beans		roni		sm Peach	
4 teasp Butter		1 sm Peach		2 h tbsp Squash		1 cup Tea	
2 slices Pineapple		1 slice Bread		4 llice Pineapple		1 slice Bread	
1 cup Tea		$3 \times \frac{1}{2} \times \frac{1}{2}$		4 Date		$3 \times \frac{1}{2} \times \frac{1}{2}$	
8 teasp Cream		1 cup Tea		1 cup Tea		8 teasp Cream	
1 teasp Sugar		$\frac{1}{2}$ glass Cream		10 teasp Sugar		5 teasp Sugar	
		5 teasp Sugar		4 teasp Butter		4 teasp Butter	
		4 teasp Butter					

## Dietary Treatment of Oxaluria

The dietary treatment of oxaluria should be regulated to avoid those foods which have a high oxalic acid content, such as

Potato	Tobacco
Cocoa	Bean
Chocolate	String Beans
Black tea	Cabbage
Coffee	Gooseberry
Sorrel	Plum
Spinach	Endive
Rhubarb	Strawberry
Fig dried	Tomato
Beets	Bread
C lery	Grapefruit

These foods should be omitted from your diet list

## Neutral and Alkali and Acid Producing Foods

### ALKALI PRODUCING FOODS

Almonds	Leeks
Apples	Lemons
Apricots	Lettuce
Asparagus	Limes
Bananas	Maple syrup
Beans all varieties	Milk
Beets	Molasses
Blackberries	Mushrooms
Buttermilk	Muskmelon
Cabbage	Olives
Cantaloupe	Oranges
Carrots	Parsnips
Cauliflower	Peaches
Celery	Pears
Chestnuts	Peas
Cherries red	Pineapple
Citron	Potatoes
Coconut fresh dried	Pumpkin
Cream	Radishes
Cucumbers	Raisins
Currents	Rhubarb
Dates	Rutabagas
Figs	Spinach
Greens salad	Strawberries
Grapefruit	Sweet potatoes
Grapes	Tomatoes
Horseradish	

Especially efficient alkali producing foods

### ACID PRODUCING FOODS

Barley pearl	Ment
Bread white	Bacon
Bread whole wheat	Beef
Cake	Fowl
Cereals breakfast	Frog
Cheese cheddar	Ham
Clams	Liver
Corn sweet dried	Mutton
Crackers	Pork
Cranberries	Rabbit
Doughnuts	Veal
Egg whole	Meat peptone
Egg white	Oatmeal
Egg yolk	Oysters
Fish fresh	Parsley
Fish smoked	Peanuts
Fish canned	Plums prunes*
Flour white	Rice
Flour whole wheat	Spaghetti
Lentils, dried	

### NEUTRAL FOODS (no ash)

Butter	Olive oil
Karo	Salad oils
Lard	Sugar
Margarine	Tapioca

The ash of these foods is alkaline but be sure they contain substances which form hippuric acid in the body they increase the acidity of the urine.

### Neutral Low Sodium Diet

- 1 Protein food unrestricted or forced
- 2 Total amount of food, calories, as indicated, low fat and butter intake
- 3 Low salt content, very sharp restriction
- 4 Medication Ammonium chloride, 30 Gm (45 gr), with each meal, in capsules (six capsules each containing 0.5 Gm [7½ gr] taken during the meal), three days on and two days off. The capsules should not be replaced by the chocolate coated or the enteric coated pills because the latter sometimes pass through the gastro intestinal tract without being absorbed. The ammonium chloride can be given in this dosage for an indefinite time without injurious effects. Patients with nephritis have taken ammonium chloride in this manner for a period of five years.
- 5 Water intake is unrestricted, although excessive quantities of liquids should not be taken
- 6 Diet Approximate neutral low sodium diet

#### GROUP A Following food may be taken daily

- 1 Egg white, meat, fish and fowl as desired
- 2 Bread as desired
- 3 Cereal, one of the following Farina oatmeal, rice, puffed rice, or puffed wheat
- 4 Potato and one or more servings of any of the following macaroni, spaghetti, rice, corn, cranberries, prunes, plums
- 5 Milk if desired
- 6 Vegetables and fruits daily serving of any fruit and of any vegetable not included in Groups "B" and "C" as desired
- 7 Butter, cream, honey, jelly, jams, sugar and candy permitted as desired
- 8 Tea and coffee as desired

#### GROUP B Foods to be avoided

All salt meats and fish or bread crackers and butter prepared with salt or carrots, clams, condensed milk, raisins, caviar, cowpeas, olives, spinach, cheese, oysters

#### GROUP C Foods to be taken no more than twice weekly

Lima beans, beets, buttermilk, cantaloupe, cauliflower, celery, chard, dried coconut, dried currants, dates, figs, horseradish, kohlrabi, limes, muskmelon, peanuts, peaches, mustard, pumpkin, radishes, rutabagas, strawberries, turnips, turnip tops, water cress

## Elimination Diets for Determining Food Allergy

## ELIMINATION DIET No 1

## CORN WHEAT, MILK, AND EGG FREE DIET

Cereal	Rice
Bread	None
Meat or fish	Lamb
Fruits jams and fruit drinks	Lemon pears peaches
Miscellaneous	Sugar, olive oil salt olives (unstuffed) maple syrup and gelatin

The rice must not be fried with butter or lard but only with olive oil

The juice from fruits listed can be used in drinks salads for desserts and for jams and sauces

Gravies for meats and sauces for vegetables can be thickened only with rice flour

No foods beverages spices condiments or flavoring other than those listed in this diet

Breakfast	Menu
Rice (natural)	Boiled served with peach or pear juice and sugar or maple syrup
Pears or peaches	Large helping fresh or canned
Drinks	Lemonade with plenty of sugar
Lunch and Dinner	
Soup	Lamb broth with rice and carrots
Salad	Lettuce with pears or peaches with olives and olive oil and lemon Green or ripe olives unstuffed
Meats	Roast lamb with gravy made with rice flour Broiled lamb chops
Vegetables	Spinach or carrots Boiled natural rice
Dessert	Lemon gelatin or pears or peaches
Drinks	Lemonade with plenty of sugar

## ELIMINATION DIET No 2

Cereals	Cornflakes cornmeal mush hominy
Bread	Corn pone
Meat	Bacon chicken
Vegetables	Squash peas artichokes asparagus
Fruits jams and fruit drinks	Pineapple apricots prunes
Miscellaneous	Sugar Mazola oil salt

Prescribed fruits can be used in drinks salads desserts jams and sauces

Gravies for meats and sauce can be thickened with cornstarch only Large servings of the above foods are to be taken

No foods beverages spices condiments or flavorings except those listed on this diet



Menu	
<i>Breakfast</i>	
Cereal	Cornflakes cornmeal mush, or hominy flakes or grits with sugar or prune juice or apricot juice
Fruit	Pineapple, apricots or prunes, fresh dried or canned
Bread	Corn pone*
Drinks	Pineapple juice with sugar if desired
Meat	Bacon
<i>Lunch and Dinner</i>	
Soup	Chicken broth
Meats	Chicken with gravy thickened with cornstarch Chicken may be roasted broiled, or fried in Mazola oil stewed or baked
Vegetables	Squash asparagus, peas or hominy Fried corn meal mush fried in Mazola oil or bacon fat
Bread	Corn pone
Dessert	Pineapple apricot or prunes
Drinks	Pineapple juice water

## ELIMINATION DIET NO 3

Cereal	Rice, tapioca
Bread	None
Meat or fish	Beef
Vegetables	Tomatoes celery and string beans
Fruits jams and fruit drinks	Grapefruit pears peaches
Miscellaneous	Sugar Wesson oil salt gelatin, maple syrup

Prescribed fruits can be used in drinks salads desserts and jams and gravies for meat and sauces can be thickened with rice flour or tapioca only

No foods beverages, spices, condiments, or flavorings other than those listed on this diet

Menu	
<i>Breakfast</i>	
Fruit	Grapefruit, dried peaches
Cereal	Rice (natural), boiled rice flakes with peaches or pear juice puffed rice, Rice Krispies
Bread	None
Drinks	Peach or pear juice with sugar if desired
<i>Lunch and Dinner</i>	
Soup	Beef broth with tomatoes and celery
Meat	Beef roasted broiled fried in Wesson oil baked or stewed
Vegetables	Tomatoes celery and string beans
Salad	Tomato or celery
Bread	None
Dessert	Tapioca baked with peaches and sugar and flavored with peach juice gelatin with peaches or pears
Drinks	Peach or pear or grapefruit juice with sugar as desired

\*Corn pone is made with cornmeal salt water and bacon fat or Mazola oil It is baked in the oven

## ELIMINATION DIET No 4

Cereals	Rice rye
Bread	Rye rice Ry Krisp
Meat	Cod halibut white fish
Vegetables	Lettuce carrots, peas beets
Fruit and jams	Pineapple pear apricots
Miscellaneous	Sugar olive oil, salt, olives ( <i>unstuffed</i> )
Beverages	Fruit juice from any of above fruits

The rice must not be fried with butter or lard but only with olive oil.

The juice from fruits listed can be used in drinks, salads for dessert and for jams and sauces

Gravies for meats and sauces for vegetables can be thickened only with rice flour

No foods beverages spices condiments or flavorings other than those listed on this diet

## Menu

<i>Breakfast</i>	
Cereal	Rice boiled with fruit juice
Fruit	Pineapple apricots pears fresh dried or canned
Bread	Rye rice Ry Krisp
Drinks	Fruit juices with sugar if desired
<i>Lunch and Dinner</i>	
Meat	Cod halibut white fish
Vegetables	Lettuce carrots peas beets
Salad	Fruit or vegetable—use those listed Serve with olive oil
Bread	1 y e rice Ry Krisp
Dessert	Pears pineapple apricots fruit rice pudding
Drinks	Fruitade of mixed fruit juices with sugar if desired

**Note** The Elimination Diets of Dr Albert Rowe of Oakland Calif, may be obtained on printed sheets from J W Stacey, Inc Flood Bldg San Francisco Calif

## A Diet for Precipitation of Food Sensitivity

## ALLERGY DIET

This diet consists of wheat milk egg potato orange sugar chocolate, and salt

## Suggested Menu

<i>Breakfast</i>	
	Milk malted m lk or Postum
	Orange o nge ju ce or orang marmalade
	Wheat cereal (bran Cream of Wheat Grape Nuts, Puffed Wheat Palston, Shredded Wheat or Wheaties)
	Bread plain or toasted
	Butt
	Butter
	C eam and sugar
	Eggs
	Waffles or gr ddle cakes

**Lunch**

Milk, malted milk, buttermilk, or Postum  
 Bread plain or toasted  
 Biscuit  
 Rolls  
 Crackers  
 Butter  
 Eggs  
 Cheese, cottage, American, or Swiss  
 Macaroni or spaghetti with cheese  
 Potato, creamed, baked, boiled, fried, or au gratin  
 Orange, orange flavored custard or pudding, orange sherbet  
 Vanilla ice cream  
 Plain pound cake or vanilla wafers

**Dinner**

Same as above  
 Milk with or without crackers between meals and at bedtime

### High Vitamin 'U' Diet for Peptic Ulcer (Modified)

**General Principles** Use daily and liberally a well balanced diet, with emphasis on fresh vegetables and juices that contain vitamin U

**Selection of Food**

**Meats** Chicken scraped beef lamb chops calves liver tender fillet steaks sweetbreads brains white fish tender lean roast beef or lamb, or bacon Avoid veal and pork Avoid meat gravies Meats should be broiled baked boiled, or lightly fried Use no salt

**Egg** Egg white only Only two or more whites daily soft cooked or poached 2 minutes

**Dairy Products** Use margarine and Miltor or Starline A quart of skimmed milk daily with or between meals Cottage or Swiss cheese or any milk processed cheese

**Vegetables** Mu krooms peas string beans carrots beets asparagus try summer squash, chopped spinach stewed tomatoes or broccoli Any others if puréed Two salads per day made from fresh lettuce watercress romaine parsley tomato avocado with olive oil dressing, and (if well tolerated) cucumber and green peppers

One half to one pint fresh cabbage or mixed vegetable juices as celery spinach and green pepper daily

**Fruits** All should be cooked or canned Peaches pears white cherries apricots without skins apples without skins juices of allowed fruits if well tolerated Orange juice or tomato juice and two ripe bananas and ripe olives should be taken daily

**Cereals** Farina Cream of Wheat rice cornmeal oatmeal cornflakes Malt Meal, puffed rice or any other cereal without bran

**\*Salad Dressing Recipe**

Lemon juice	$\frac{3}{4}$ cup
Olive oil	$1\frac{1}{2}$ cup
Salt	~ teaspoonfuls
Sugar	2 table spoonfuls

Potatoes	White potatoes—baked mashed boiled creamed or escalloped Avoid fried potatoes Plain paghetti noodle macaroni, or rice may be used as substitute
Bread	Toast white or graham bread plain crackers waffles hot cakes
Desserts	Simple puddings such as cornstarch pudding junket custard tapioca rice pudding plain white cake sponge cake white cookies Avoid rich pies pastrie and rich desserts
Sweets	Sugar honey maple or Karo syrup clear jelly in moderation Avoid rich jams or marmalades containing rind or seeds.
Soups	Cream soups made from vegetables listed above oyster stew bouillon consommé and clam broth if desired
Nourishments	10 A.M. 3 P.M. and 8 P.M. skimmed milk fresh vegetable juice or eggnog

## Menu

## Breakfast

Fruit	-	One serving
Cereal (if desired)	-	One serving
Eggs cooked (whites only)	-	Two
Bread or toast (if desired)	-	One slice
Margarine	-	One pat
Beverage—skimmed milk cocoa chocolate coffee with cream	-	One cup

## 10 A.M.

Skimmed milk thickened with Starlac or Protinum	One glass
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## Dinner

Soup	-	If desired
Meat	-	One medium serving
Potato or substitute (if desired)	-	One serving
Vegetable	-	One serving
Salad with special olive oil dressing	-	One serving
Bread (if desired)	-	One slice
Margarine	-	One pat
Dessert—bananas	-	One serving
Fresh vegetable juice	-	One glass

## 3 P.M.

Skimmed milk	One glass
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## Supper

Soup	-	If desired
Meat	-	One medium serving
Vegetable	-	One serving
Salad (coleslaw with ripe olives)	-	One serving
Bread	-	One slice
Margarine	-	One pat
Dessert (if desired)	-	One serving
Fresh cabbage juice	-	One glass

Eggnog (3 or more times a week with or between meals)

Egg whites	1	teaspoonful
Sugar	1	teaspoonful
Vanilla	1	teaspoonful
Mint or S. ya	1	cup
Skimmed milk	1	pat

*Lunch*

Milk, malted milk buttermilk, or Postum  
 Bread, plain or toasted  
 Biscuit  
 Rolls  
 Crackers  
 Butter  
 Eggs  
 Cheese, cottage, American or Swiss  
 Macaroni or spaghetti with cheese  
 Potato, creamed, baked, boiled, fried, or au gratin  
 Orange, orange flavored custard or pudding, orange sherbet  
 Vanilla ice cream  
 Plain pound cake or vanilla wafers

*Dinner*

Same as above  
 Milk with or without crackers between meals and at bedtime

### High Vitamin 'U' Diet for Peptic Ulcer (Modified)

**General Principles** Use daily and liberally a well balanced diet with emphasis on fresh vegetables and juices that contain 'vitamin U'

**Selection of Food**

**Meats** Chicken scraped beef lamb chops calves liver tender fillet steaks, sweetbreads, brains, white fish tender lean roast beef or lamb or bacon Avoid veal and pork Avoid meat gravies Meats should be broiled baked boiled, or lightly fried Use no salt

**Egg** Egg white only Only two or more whites daily soft cooked or poached 2 minutes

**Dairy Products** Use margarine and Milnot or Starlac A quart of skimmed milk daily with or between meals Cottage or Swiss cheese or any milk processed cheese

**Vegetables** Mushroom, peas string bean carrots beets a paragus tips summer squash chopped spinach stewed tomatoes or broccoli Any others if puréed Two salads per day made from fresh lettuce watercress romaine parsley tomato avocado with olive oil dressing,\* and (if well tolerated) cucumber and green peppers

One half to one pint fresh cabbage or mixed vegetable juices as celery spinach and green pepper daily

**Fruits** All should be cooked or canned Peaches pears white cherries apricots without skins apples without skins, juices of allowed fruits if well tolerated Orange juice or tomato juice and two ripe bananas and ripe olives should be taken daily

**Cereals** Farina Cream of Wheat rice cornmeal oatmeal cornflake, Malt o Malt puffed rice or any other cereal without bran

**\*Salad Dressing Recipe**

Lemon juice	$\frac{3}{4}$ cup
Olive oil	$1\frac{1}{2}$ cup
Salt	2 teaspoonfuls
Sugar	2 tablespoonfuls

## Low Cholesterol and Fat Recipes

By MRS SETH I FWIS

## 1. Ham Cured Smoked Beef Roast

Any meat plant can process beef identically as they do pork for ham and the result is palatable and delicious. It gives one an opportunity to enjoy something real nice in cold sliced meat.

## Macaroni Dishes

In order to serve baked macaroni I substitute grated Swiss cheese for yellow cheese and use oleo very sparingly. Prepare according to usual recipe that is a layer of boiled drained macaroni then seasoning a little oleo and grated cheese, then alternate this to the top of the dish topping with cheese. Bake in 350 oven for 40 minutes or an hour until browned.

Grated Swiss cheese over spaghetti which has been boiled and to which a small piece of oleo has been added makes a palatable dish and to vary this use tomato sauce over spaghetti and then top with Swiss cheese.

## 3 White Salad Sauce made with Starlac

1 t. oleo	$\frac{1}{2}$ T grated onion
1 T flour	Salt and pepper
1 $\frac{1}{4}$ cups prepared Starlac	Chopped parsley

Melt oleo add flour and stir until smooth add seasonings then gradually add milk and stir until thickened. Add cubed roast chicken, or boiled shrimp or crabmeat. Serve in toast cups. This sauce is really enough for three servings and is also nice on certain vegetables when grated Swiss cheese has been added to it.

## 4 Baked Brown Bread

1 $\frac{1}{2}$ c graham flour	$\frac{1}{2}$ t salt
1 $\frac{1}{2}$ c yellow corn meal	" t soda
" c white flour	1 c raisins
$\frac{1}{2}$ t double acting baking powder	1 c molasses
or 1 t single acting baking powder	1 c sour milk or buttermilk

Sift flour before measuring. Add salt, soda and baking powder and sift again. Add graham flour and corn meal. Blend thoroughly. Add raisins then molasses and stir well.

Line two small pans with wax paper divide mixture and bake 50 to 60 min at 350.

5 Cauliflower, asparagus and other vegetable variations. Melt Swiss cheese in Starlac and serve it over them as a sauce.

## 6 Potatoes Au Gratin

Creamed potatoes (cooked) about 2 cups  
1 t finely chopped parsley  
1 c grated Swiss cheese

Whip half of cheese into potatoes and add parsley. Turn into baking dish sprinkle with cheese. Bake at 350 for 30 min.

Sometimes for a change chop Canadian bacon which has been cooked dry and drained well and whip it into the potato mixture.

9 P M (Bedtime)

Milk -----	One glass
Banana -----	One

**Notes on Vitamin U Diet for Peptic Ulcer**—Dr Frederick W Kroll has found after a great deal of experimentation, that using 50 % of the raw, unpasteurized cabbage juice and the other 50 % composed of approximately equal volumes of celery juice, carrot juice, green pepper juice, and spinach juice makes the entire mixture a much more palatable drink. He advises the patient drinking a full quart of the mixture a day. Before drinking it is wise to strain the juice through a fairly coarse tea strainer because frequently considerable raw cellulous pulp comes through from the juicer, and if it is drunk, while it will not hurt the patient it may cause a great deal of gas which is quite annoying.

It has been found much more satisfactory to grind up the vegetables twice daily, in the morning and at noon, or in the morning and before the evening meal. This is better than attempting to grind up the whole quart at one time. The reason for this is that the fresher the juice, the better it tastes and more of the anti ulcer factor is obtained. As the juice stands even in the refrigerator, the anti ulcer factor is slowly dissipated and is found to be virtually nonexistent at the end of twenty four hours. Therefore, the reason for grinding as freshly as possible. In drinking the juice, Kroll has found it best to take small sips of it frequently with the regular meals or at other times between meals with a few crackers or Triscuits. Juice that is not used at the time it is obtained should be stored in the refrigerator. Naturally, the patients are advised to eliminate any of the vegetables in the list that disagree with them.

The "Health Way Juice Extractor" manufactured by the Stomach Manufacturing Co, 31 East Montecito Ave Sierra Madre, Calif, is recommended. Vegetables that have been found by Dr Garnett Cheney to contain the anti ulcer factor are cabbage celery carrots cucumbers green peppers, spinach, and perhaps tomatoes. Juices from these vegetables have been found effective in healing ulcers in patients on otherwise regular balanced diet. The only fruit that contains the anti ulcer factor is the ripe raw banana. Each patient is advised to eat as many of them as he can, provided that they do not disagree with him.

#### References

- Cheney, G. California Med 70 1, 1949  
 Kroll, F W. Personal communication

Add sugar and salt to milk, stir until dissolved. Pour over egg whites and blend. Add flavorings. Pour into custard cups. Bake in pan of hot water in moderate oven at 350° about 30 to 40 min or until mixture does not stick to knife. Serves 4 or 5.

**Strawberry Whip**      $\frac{3}{4}$  cup frozen strawberries defrosted or fresh (or peaches, apricots, grated apple, other fruits)  
                                  1 egg white  
                                   $\frac{1}{2}$  c sugar  
                                   $\frac{1}{2}$  T lime juice or lemon juice

Combine ingredients and whip in an electric mixer, 20 min or longer until mixture is stiff. Serve with white custard.

**Fruit Cups**     1 can pie cherries or 2 c fruit     2 T cornstarch  
                          1 c sugar      $\frac{1}{4}$  c water

I prepare cherries as for pie filling that is add sugar to cherries and boil. Add cornstarch which has been dissolved in water and continue to cook mixture over a low fire until clear. Pour into custard cups or other baking dishes and meringue plates in 350° oven and brown. Serve cold.

To vary use different fruits such as crushed pineapple, mashed canned apricot or fresh peaches prepared as above.

These fruit mixtures too can be used between layers of angel food cake in large baking dish or casserole with meringue on top and slightly browned.

**Lime Mint Delight** This dessert is inexpensive and relatively low in calories. It is easy to make and is cool and refreshing in color and taste.

1 pkg lime flavored gelatin      $1\frac{1}{2}$  c sugar  
 1 c boiling water     1 qt Starlac milk  
 Juice of 2 lemons     Fresh mint

Dissolve gelatin in boiling water. Cool. Add lemon juice and sugar and stir until sugar is dissolved. Cool thoroughly in refrigerator but do not let mixture congeal. Remove and add milk which is also well chilled. Beat with rotary egg beater until well blended. Pour into freezing tray and freeze until firm stirring each 20 min. Serve in dessert dishes topped with lightly crushed fresh mint. Serves 6 (From *July American Girl*).

**Easy Pineapple Sherbet** The crushed pineapple makes this different from sherbet.  
 1 14 oz can crushed pineapple      $\frac{2}{3}$  c granulated sugar  
 1 T lemon juice     1 pint milk (Starlac prepared)  
 1 t grated lemon rind

Combine pineapple, lemon juice and rind and sugar. Add milk and stir until sugar is dissolved. Turn into refrigerator tray and stir occasionally until nearly firm. Remove to bowl, break up chunks and beat until light and creamy but not melted. Return to tray for final freezing. Serves 4 or 5.

**Meringues**     2 egg whites      $\frac{1}{2}$  t water  
                           $\frac{1}{4}$  c fine granulated sugar     3 drops vinegar  
                           $\frac{1}{2}$  t vanilla     Pinch of salt

Beat whites until stiff, add vinegar, water and salt. Beat a little then add sugar gradually and finally add vanilla. Beat until stiff but not dry. Drop spoonfuls of meringue on greased cookie sheet and cook for one hour in 350° oven. Cool slowly. Serve filled with fresh fruit or white custard topped with chocolate sauce or preserved fruit.



7 *Scalloped Potatoes*

Slice about 6 raw potatoes cover the bottom of a baking dish with thin slices of raw potato. Put over this a light dredging of flour, salt, pepper a little grated onion and chopped parsley. Alternate potatoes and seasonings until dish is full. Pour prepared Starlac to cover and bake in moderate oven at 350° for one hour.

When seasoning potatoes, a few dots of oleo along with the seasonings or a little grated Swiss cheese improves the dish.

8 *Sweet Potatoes—Candied*

Cook  $1\frac{1}{2}$  cups of sugar,  $\frac{1}{4}$  cup of water, and 1 whole sliced orange or a small can of crushed pineapple, to a medium syrup. Parboil about 5 small potatoes or yams and pare and cut in halves lengthwise. Add these to syrup and continue cooking slowly until syrup is very thick. Serve with syrup and orange slices or fruit over potatoes.

*Pineapple Marshmallow Sweet Potatoes*

2 c mashed sweet potatoes	1 c diced pineapple
1 c prepared Starlac milk	marshmallows
1 c pineapple juice or orange juice	

Thoroughly mix all the ingredients and heat until light and fluffy. Use more milk or fruit juice, if needed. Place in a casserole and bake until heated through. Remove from oven and cover the top with marshmallows. Return to oven to brown. Serve with poultry or roast.

9 *Baked Apples*

When one cannot eat cream this is nice for a change. Before baking apples peel them halfway down, then bake. When apples are tender and syrup has cooked down take them out of oven fill centers with raisins, chopped cherries or crushed pineapple. Top with meringue and return to the oven to brown. Remove to dessert dishes and spoon jelly or syrup from pan in which apples were baked over them.

10 *Suggestions for Low Cholesterol Desserts*

<i>Chocolate Custard</i>	1 $\frac{1}{2}$ c sugar	$\frac{1}{2}$ c prepared Starlac
	$\frac{1}{2}$ P corn starch	$\frac{1}{2}$ T cocoa

Mix sugar, cornstarch and cocoa; gradually add milk. Cook over low fire constantly stirring until mixture thickens. Remove from fire and continue stirring until smooth and add two teaspoonfuls of vanilla extract. This mixture can be poured into custard cups and topped with meringue. Place in oven at 325° until meringue is light brown. It can be used as sauce over slices of angel food cake too or alternating layers of the cake and custard topped with meringue and baked until brown.

*Meringue for Topping*    2 egg whites stiffly beaten  
    3 T sugar

Add sugar gradually to beaten egg white. Spoon on dessert and place in oven at 325° and bake to light brown.

<i>Snow White Custard</i>	$\frac{1}{4}$ c sugar	3 slightly beaten egg whites
	Few grains salt	$\frac{1}{2}$ t vanilla
	$1\frac{1}{4}$ c milk scalded	$\frac{1}{4}$ t almond extract
	(prepared Starlac)	

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